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National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection Byproducts Rule; National Primary and Secondary Drinking Water Regulations: Approval of Analytical Methods for Chemical Contaminants; Proposed Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Parts 141, 142 and 143

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National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection Byproducts Rule; National Primary and Secondary Drinking Water Regulations: Approval of Analytical Methods for Chemical Contaminants

AGENCY: Environmental Protection Agency.

ACTION: Proposed rule.

SUMMARY: In this document, the Environmental Protection Agency (EPA) is proposing maximum contaminant level goals (MCLGs) for chloroform, monochloroacetic acid (MCAA) and trichloroacetic acid (TCAA); National Primary Drinking Water Regulations (NPDWRs) which consist of maximum contaminant levels (MCLs) and monitoring, reporting, and public notification requirements for total trihalomethanes (TTHM—a sum of chloroform, bromodichloromethane, dibromochloromethane, and bromoform) and haloacetic acids (HAA5—a sum of mono-, di-, and trichloroacetic acids and mono- and dibromoacetic acids); and revisions to the reduced monitoring requirements for bromate. This document also specifies the best available technologies (BATs) for the proposed MCLs. EPA is also proposing additional analytical methods for the determination of disinfectants and disinfection byproducts (DBPs) in drinking water and proposing to extend approval of DBP methods for the determination of additional chemical contaminants. This set of regulations proposed today is known as the Stage 2 Disinfectants and Disinfection Byproducts Rule (Stage 2 DBPR). EPA's objective for the Stage 2 DBPR is to reduce the potential risks of reproductive and developmental health effects and cancer associated with disinfection byproducts (DBPs) by reducing peak and average levels of DBPs in drinking water supplies.

The Stage 2 DBPR applies to public water systems (PWS) that are community water systems (CWSs) or nontransient noncommunity water systems (NTNCWs) that add a primary or residual disinfectant other than ultraviolet light or deliver water that has been treated with a primary or residual disinfectant other than ultraviolet light.

DATES: The Agency requests comments on today's proposal. Comments must be received or post-marked by midnight November 17, 2003.

ADDRESSES: Comments may be submitted by mail to: Water Docket, Environmental Protection Agency, Mail Code 4101T, 1200 Pennsylvania Ave., NW., Washington, DC 20460, Attention Docket ID No. OW–2002–0043. Comments may also be submitted electronically or through hand delivery/courier by following the detailed instructions as provided in section I.C. of the **SUPPLEMENTARY INFORMATION** section.

FOR FURTHER INFORMATION CONTACT: For technical inquiries, contact Tom Grubbs, Office of Ground Water and Drinking Water (MC 4607M), U.S. Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone (202) 564–5262. For regulatory inquiries, contact Jennifer McLain at the same address; telephone (202) 564–5248. For general information contact the Safe Drinking Water Hotline, Telephone (800) 426–4791. The Safe Drinking Water Hotline is open Monday through Friday, excluding legal holidays, from 9 a.m. to 5:30 p.m. Eastern Time.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Who Is Regulated by This Action?

Entities potentially regulated by the Stage 2 DBPR are community and nontransient noncommunity water systems that add a primary or residual disinfectant other than ultraviolet light or deliver water that has been treated with a primary or residual disinfectant other than ultraviolet light. Regulated categories and entities are identified in the following chart.

Category	Examples of regulated entities
Industry	Community and nontransient noncommunity water systems that add a primary or residual disinfectant other than ultraviolet light or deliver water that has been treated with a primary or residual disinfectant other than ultraviolet light.
State, Local, Tribal, or Federal Governments	Community and nontransient noncommunity water systems that add a primary or residual disinfectant other than ultraviolet light or deliver water that has been treated with a primary or residual disinfectant other than ultraviolet light.

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be regulated by this action. This table lists the types of entities of which EPA is now aware that could potentially be regulated by this action. Other types of entities not listed in this table could also be regulated. To determine whether your facility is regulated by this action, you should carefully examine the definition of ''public water system'' in § 141.2 and the section entitled ''coverage'' (§ 141.3) in Title 40 of the Code of Federal Regulations and applicability criteria in § 141.600 and 141.620 of today's proposal. If you have questions regarding the applicability of the Stage 2 DBPR to a particular entity, contact one of the persons listed in the preceding section entitled **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Copies of This Document and Other Related Information?

1. *Docket.* EPA has established an official public docket for this action under Docket ID No. OW–2002–0043. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Water Docket in the EPA Docket Center, (EPA/DC) EPA West, Room B102, 1301 Constitution Ave., NW., Washington, DC. The EPA Docket Center Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the Water Docket is (202) 566–2426. For access to docket material, please call (202) 566–2426 to schedule an appointment.

2. *Electronic Access.* You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/.*

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Once in the system, select ''search,'' then key in the appropriate docket identification number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in section I.B.1.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the Docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and to Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket identification number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments.

1. *Electronically.* If you submit an electronic comment as prescribed below, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

a. *EPA Dockets.* Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket,* and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in Docket ID No. OW–2002–0043. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

b. *E-mail.* Comments may be sent by electronic mail (e-mail) to *OW-Docket@epa.gov,* Attention Docket ID No. OW–2002–0043. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the Docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

c. *Disk or CD ROM.* You may submit comments on a disk or CD ROM that you mail to the mailing address identified in section I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By Mail.* Send three copies of your comments and any enclosures to: Water Docket, Environmental Protection Agency, Mail Code 4101T, 1200 Pennsylvania Ave., NW., Washington, DC 20460, Attention Docket ID No. OW–2002–0043.

3. *By Hand Delivery or Courier.* Deliver your comments to: Water Docket, EPA Docket Center, Environmental Protection Agency, Room B102, 1301 Constitution Ave., NW., Washington, DC, Attention Docket ID No. OW–2002–0043. Such deliveries are only accepted during the Docket's normal hours of operation as identified in section I.B.1.

D. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at your estimate.

5. Provide specific examples to illustrate your concerns.

6. Offer alternatives.

7. Make sure to submit your comments by the comment period identified.

8. To ensure proper receipt by EPA, identify the appropriate docket identification number in the subject line on the first page of your response. It would also be helpful if you provided the name, date, and **Federal Register** citation related to your comments.

Abbreviations Used in This Document

AIPC All Indian Pueblo Council
ALT Alanine aminotransferase
AST Aspartate aminotransferase
ASTM American Society for Testing and Materials
AWWA American Water Works Association
AwwaRF American Water Works Association Research Foundation
BAT Best available technology
BCAA Bromochloroacetic acid

BDCM Bromodichloromethane
CWS Community water system
DBAA Dibromoacetic acid
DBCM Dibromochloromethane
DBP Disinfection byproduct
DBPR Disinfectants and Disinfection Byproducts Rule
DCAA Dichloroacetic acid
DOC Dissolved organic carbon
EA Economic analysis
EC Enhanced coagulation
EDA Ethylenediamine
ED_{10} Maximum likelihood estimate of a dose producing effects in 10 percent of animals
EPA United States Environmental Protection Agency
FACA Federal Advisory Committee Act
FBRR Filter Backwash Recycling Rule
GAC Granular activated carbon
GC/ECD Gas chromatography using electron capture detection
GWUDI Ground water under the direct influence of surface water
HAA5 Haloacetic acids (five) (sum of monochloroacetic acid, dichloroacetic acid, trichloroacetic acid, monobromoacetic acid, and dibromoacetic acid)
IC Ion chromatography
ICR Information Collection Request
IC/ICP–MS Ion chromatograph—coupled to an inductively coupled plasma mass spectrometer
IDSE Initial distribution system evaluation
ILSI International Life Sciences Institute
IESWTR Interim Enhanced Surface Water Treatment Rule
IPCS International Programme on Chemical Safety
IRIS Integrated Risk Information System (EPA)
kWh/yr Kilowatt hours per year
LED_{10} Lower 95 percent confidence bound of the maximum likelihood estimate of the dose producing effects in 10 percent of animals
LH Luteinizing hormone
LOAEL Lowest observed adverse effect level
LRAA Locational running annual average
LT1ESWTR Long Term 1 Enhanced Surface Water Treatment Rule
LT2ESWTR Long Term 2 Enhanced Surface Water Treatment Rule
MBAA Monobromoacetic acid
MCAA Monochloroacetic acid
MCL Maximum contaminant level
MCLG Maximum contaminant level goal
M-DBP Microbial and disinfection byproducts
mg/L Milligram per liter
MRL Minimum reporting level
MRDL Maximum residual disinfectant level
MRDLG Maximum residual disinfectant level goal
MTBE Methyl tertiary butyl ether
mWh Megawatt-hours
NATICH National Air Toxics Information Clearinghouse
NDIR Nondispersive infrared detection
NDMA N-nitrosodimethylamine
NDWAC National Drinking Water Advisory Council
NF Nanofiltration
NOAEL No Observed Adverse Effect Level
NODA Notice of data availability
NPDWR National primary drinking water regulation
NRWA National Rural Water Association
NTNCWS Nontransient noncommunity water system
NTP National Toxicology Program
NTTAA National Technology Transfer and Advancement Act
ODA o-dianisidine dihydrochloride
OMB Office of Management and Budget
OSTP Office of Science and Technology Policy
PAR Population attributable risk
PE Performance evaluation
PWS Public water system
QC Quality control
RAA Running annual average
RFA Regulatory Flexibility Act
RfD Reference dose
RSC Relative source contribution
RSD Relative standard deviation
SAB Science Advisory Board
SAC Selective anion concentration
SBAR Small Business Advisory Review
SBREFA Small Business Regulatory Enforcement Fairness Act
SDWA Safe Drinking Water Act, or the ''Act,'' as amended in 1996
SER Small Entity Representative
SGA Small for gestational age
SUVA Specific ultraviolet absorbance
SWAT Surface Water Analytical Tool
SWTR Surface Water Treatment Rule
TAME Tertiary amyl methyl ether
TCAA Trichloroacetic acid
TCR Total Coliform Rule
THM Trihalomethane
TOC Total organic carbon
TTHM Total trihalomethanes (sum of four THMs: chloroform, bromodichloromethane, dibromochloromethane, and bromoform)
TWG Technical work group
UMRA Unfunded Mandates Reform Act
USDOE EIA U.S. Department of Energy, Energy Information Administration
UV 254 Ultraviolet absorption at 254 nm
WTP Willingness To Pay

Table of Contents

I. Summary

A. Why Is EPA Proposing the Stage 2 DBPR?

The Environmental Protection Agency is committed to ensuring that all public water systems provide clean and safe drinking water. Disinfectants are often an essential element of drinking water treatment because of the barrier they provide against harmful waterborne microbial pathogens. However, disinfectants react with naturally occurring organic and inorganic matter in source water and distribution systems to form disinfection byproducts (DBPs) that may pose health risks. The Agency is proposing the Stage 2 Disinfectants and Disinfection Byproduct Rule (DBPR) to reduce potential cancer, reproductive, and developmental risks from DBPs.

The Stage 2 DBPR augments the Stage 1 DBPR that was finalized in 1998. The proposed Stage 2 DBPR focuses on monitoring and reducing concentrations of two classes of DBPs: total trihalomethanes (TTHM) and haloacetic acids (HAA5). In part, these two groups of DBPs are used as indicators of the various byproducts that are present in disinfected water. This means that concentrations of TTHM and HAA5 are monitored for compliance, but their presence in drinking water is representative of many other DBPs that may also be present in the water; likewise, a reduction in TTHM and HAA5 indicates a reduction of total DBPs.

The Stage 2 DBPR is designed to reduce the level of exposure from disinfectants and DBPs without undermining the control of microbial pathogens. The Long Term 2 Enhanced Surface Water Treatment Rule (LT2ESWTR) will be finalized and implemented simultaneously with the Stage 2 DBPR to ensure that drinking water is microbiologically safe at the limits set for disinfectants and DBPs.

New information on health effects, occurrence, and treatment has become available since the Stage 1 DBPR, which supports the need for the Stage 2 DBPR. Several reproductive and developmental studies have recently become available, and EPA has completed a more extensive analysis of reproductive and developmental effects associated with DBPs since the Stage 1 DBPR. Both human epidemiology studies and animal toxicology studies have shown associations between chlorinated drinking water and reproductive and developmental endpoints such as spontaneous abortion, stillbirth, neural tube defects, pre-term delivery, intrauterine growth retardation, and low birth weight. New epidemiology and toxicology studies evaluating bladder and rectal cancers have also increased the weight of evidence linking these health effects to DBP exposure. The large number of people (254 million Americans) exposed to DBPs and the identified potential cancer, reproductive, and developmental risks played a significant role in EPA's decision to move forward with regulatory changes that target lowering DBP exposures beyond the requirements of the Stage 1 DBPR.

While the Stage 1 DBPR provided a major reduction in DBP exposure, new national survey data suggest that some customers are receiving drinking water with elevated, or peak DBP concentrations even when their distribution systems are in compliance with the Stage 1 DBPR. Some of these peak concentrations can be substantially greater than the Stage 1 DBPR maximum contaminant levels (MCLs). The new survey results also showed that Stage 1 DBPR monitoring sites may not be representative of peak DBP concentrations that occur in distribution systems. In addition, the new information indicates that cost-effective technologies including ultraviolet light (UV) and granular activated carbon (GAC) may be very effective at lowering DBP levels. EPA's analysis of this new information concludes that significant public health benefits may be achieved through further cost-effective reduction of DBPs in distribution systems.

Congress required EPA to promulgate the Stage 2 DBPR as part of the 1996 Safe Drinking Water Act (SDWA) Amendments (section 1412(b)(2)(C)). Today's proposal reflects consensus recommendations from the Stage 2 Microbial/Disinfection Byproducts (M-DBP) Federal Advisory Committee (the Advisory Committee). These recommendations are set forth in the M-DBP Agreement in Principle (USEPA 2000g), which can be accessed on the edocket Web site (*www.epa.gov/edocket*).

After considering the new occurrence and health effects data and analyses, EPA has determined that there is an opportunity to further reduce potential risks from DBPs. The Stage 2 DBPR being proposed today presents a cost-effective, risk targeting approach to reduce risks from DBPs. The new requirements provide for more consistent protection from DBPs across the entire distribution system and the reduction of DBP peaks. New risk targeting provisions require only those systems with the greatest risk to make capital improvements. The Stage 2 DBPR, in conjunction with the LT2ESWTR, will help public water systems deliver safer water to Americans with the benefits of disinfection to control pathogens but with fewer risks from DBPs.

B. What Does the Stage 2 DBPR Require?

The Stage 2 DBPR applies to community or nontransient noncommunity water systems that add a primary or residual disinfectant other than ultraviolet light or deliver water that has been treated with a primary or residual disinfectant other than ultraviolet light. The TTHM and HAA5 MCL values will remain the same as in the Stage 1 DBPR, although compliance calculations will be different. The proposed Stage 2 DBPR includes new MCLGs for chloroform, monochloroacetic acid, and trichloroacetic acid, but these new MCLGs do not affect the MCLs for TTHM or HAA5.

The risk targeting components of the Stage 2 DBPR will focus the greatest amount of change where the greatest amount of risk may exist. The provisions of the Stage 2 DBPR focus on identifying and reducing exposure by reducing DBP peaks in distribution systems. The first provision, designed to address significant variations in exposure, is the Initial Distribution System Evaluation (IDSE). The purpose

of the IDSE is to identify Stage 2 DBPR compliance monitoring sites for capturing peaks. Because Stage 2 DBPR compliance will be determined at these new monitoring sites, distribution systems that identify elevated concentrations of TTHM and HAA5 will need to make treatment or process changes to bring the system into compliance with the Stage 2 DBPR. By identifying compliance monitoring sites with elevated concentrations of TTHM and HAA5, the IDSE will offer increased assurance that MCLs are being met across the distribution system. Both treatment changes and awareness of TTHM and HAA5 levels resulting from the IDSE will allow systems to better control for distribution system peaks.

The IDSE is designed to offer flexibility to public water systems. The IDSE requires TTHM and HAA5 monitoring for one year on a regular schedule that is determined by source water type and system size. Systems have the option of performing a site-specific study based on historical data, water distribution system models, or other data; and waivers are available under certain circumstances. The proposed IDSE requirements are discussed in sections V.H., V.I., and V.J. of this preamble and in subpart U of the proposed rule.

The second provision of the Stage 2 DBPR, which is designed to address variations in temporal and spatial exposure, is the new compliance calculation of the MCLs. The Stage 1 DBPR running annual average (RAA) calculation allows some locations within a distribution system to have higher DBP annual averages than others as long as the system-wide average is below the MCL. The Stage 2 DBPR will base compliance on a locational running annual average (LRAA) calculation where the annual average at each sampling location in the distribution system will be used to determine compliance with the MCLs. The LRAA will reduce exposures to peak DBP concentrations by ensuring that each monitoring site is in compliance with the MCLs as an annual average, and it will provide all customers drinking water that more consistently meets the MCLs.

EPA is proposing that systems comply with the Stage 2 DBPR MCLs in two phases, designated as Stage 2A and Stage 2B. In Stage 2A, beginning three years after the rule is final, all systems must comply with MCLs of 0.120 mg/L for TTHM and 0.100 mg/L for HAA5 as LRAAs at Stage 1 DBPR sampling sites, in addition to continuing to comply with the Stage 1 DBPR MCLs of 0.080 mg/L and 0.060 mg/L as RAAs for TTHM and HAA5, respectively. In Stage 2B, systems must comply with MCLs of 0.080 mg/L and 0.060 mg/L as LRAAs for TTHM and HAA5, respectively, based on sampling sites identified through the IDSE. A more detailed discussion of the proposed Stage 2 DBPR MCL requirements can be found in sections V.D., V.I., and V.J. of this preamble and in § 141.64(b)(2) and (3), and § 141.136, and subpart V of the rule language.

The IDSE and LRAA calculation will lead to overall reductions in DBP concentrations and reduce short term exposures to high DBP concentrations, but even with this strengthened approach to regulating DBPs it will be possible for individual DBP samples to exceed the MCLs when systems are in compliance with the Stage 2 DBPR. The Stage 2 DBPR requires systems that experience significant excursions to evaluate distribution system operational practices and identify opportunities to reduce DBP concentrations in the distribution system. This provision will curtail peaks and reduce exposure to high DBP levels. Significant excursions are discussed in greater detail in section V.E.

The Stage 2 DBPR also contains provisions for regulating consecutive systems, defined in the Stage 2 DBPR as public water systems that buy or otherwise receive some or all of their finished water from another public water system on a regular basis. Uniform regulation of consecutive systems provided by the Stage 2 DBPR will ensure that consecutive systems deliver drinking water that meets applicable DBP standards. More information on regulation of consecutive systems can be found in sections V.C., V.H., V.I. and V.J.

Today's document proposes plant-based monitoring requirements for non-consecutive systems and certain consecutive systems. Plant-based monitoring means that the number of compliance monitoring locations within a distribution system is based on the number of plants, population served, and type of source water used by the distribution system. EPA is proposing population-based monitoring for consecutive systems that buy all their finished water from other public water systems. EPA is also requesting comment on whether this approach should be extended to all systems covered by today's rule. Under a population-based monitoring structure, the number of compliance monitoring locations is based only on the population served and source water type. Section V.I. describes population-based monitoring and how it might affect systems complying with this rule.

C. What Are the Economic Impacts of the Stage 2 DBPR?

EPA quantified the potential benefits of the Stage 2 DBPR by estimating the reduction in bladder cancer cases that may result from the decrease in average DBP concentrations in disinfected water. Estimated reductions in DBP-related bladder cancers (including both fatal and non-fatal cases) result in annualized benefits ranging from $0 to $986 million (using a three percent discount rate), depending on the risk level assumed.

There may also be a number of important nonquantifiable benefits associated with reducing DBPs in drinking water, the primary ones being reduced potential risk of adverse reproductive and developmental effects including miscarriage, stillbirth, neural tube defects, heart defects, and cleft palate. Although a number of studies have found an association between reproductive and developmental endpoints and short-term exposure to elevated DBP levels, a causal link has not yet been established and information is not yet available to quantify potential effects. As a result, the Agency has not included an estimate of the potential benefits from reducing reproductive and developmental risks in its primary economic impact analysis of the Stage 2 DBPR. However, an illustrative calculation of potential fetal loss risk is discussed in Section VII and presented in more detail in the Economic Analysis (USEPA 2003i) to illustrate the benefits that could be associated with this rule. Reduction in other cancers potentially associated with DBP exposure represent additional unquantified health benefits.

EPA estimates the total annualized costs of the Stage 2 DBPR to be $54 to $64 million. This estimate includes costs associated with treatment changes, the Initial Distribution System Evaluation, changes in compliance monitoring, and rule implementation activities for both public water systems and States. EPA estimates that approximately 2.8 percent of all plants will need to convert to chloramines or add advanced treatment to comply with the Stage 2 DBPR.

Table I–1 presents the estimated quantified and unquantified benefits of the Stage 2 DBPR and the estimated costs. Analyses of unquantified benefits suggest that the total benefits associated with the Stage 2 DBPR might be much greater than these estimates. By targeting risks and building on the solid foundation of the Stage 1 DBPR, the

Stage 2 DBPR will deliver cost-effective reductions in DBP levels and associated potential public health risks.

TABLE I–1.—COSTS AND BENEFITS OF THE STAGE 2 DBPR BASED ON ANNUALIZATION DISCOUNT RATE OF 3%

Costs	Benefits	Unquantified benefits
$54–64 M	$0–986 M	Reduction in potential reproductive and developmental health effects, potential reduction in colon and rectal cancer, improved taste and odor of drinking water, control of contaminants that may be regulated in the future.

II. Background

A combination of factors have influenced the development of the proposed Stage 2 DBPR. These include the initial 1992–1994 Microbial and Disinfection Byproduct (M–DBP) stakeholder deliberations and EPA's Stage 1 DBPR proposal; the 1996 Safe Drinking Water Act (SDWA) Amendments; the 1996 Information Collection Rule; the 1998 Stage 1 DBPR; other new data, research, and analysis on disinfection byproduct (DBP) occurrence, treatment, and health effects since the Stage 1 DBPR; and the Stage 2 DBPR Microbial and Disinfection Byproducts Federal Advisory Committee. The following shows how EPA arrived at this proposal for regulating disinfection byproducts.

A. What Is the Statutory Authority for the Stage 2 DBPR?

The SDWA, as amended in 1996, authorizes EPA to promulgate a national primary drinking water regulation (NPDWR) and publish a maximum contaminant level goal (MCLG) for contaminants the Administrator determines ''may have an adverse effect on the health of persons,'' is ''known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern,'' and for which ''in the sole judgement of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems'' (SDWA section 1412(b)(1)(A)). MCLGs are non-enforceable health goals set at a level at which ''no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety''. These health goals are published at the same time as the NPDWR (sections 1412(b)(4) and 1412(a)(3)).

The Agency may also consider additional health risks from other contaminants and establish an MCL ''at a level other than the feasible level, if the technology, treatment techniques, and other means used to determine the feasible level would result in an increase in the health risk from drinking water by—(i) increasing the concentration of other contaminants in drinking water; or (ii) interfering with the efficacy of drinking water treatment techniques or processes that are used to comply with other national primary drinking water regulations'' (section 1412(b)(5)(A)). When establishing an MCL or treatment technique under this authority, ''the level or levels of treatment techniques shall minimize the overall risk of adverse health effects by balancing the risk from the contaminant and the risk from other contaminants the concentrations of which may be affected by the use of a treatment technique or process that would be employed to attain the MCL or levels'' (section 1412(b)(5)(B)).

Finally, section 1412(b)(2)(C) of the Act requires EPA to promulgate a Stage 2 DBPR 18 months after promulgation of the Long Term 1 Enhanced Surface Water Treatment Rule (LT1ESWTR). Consistent with statutory requirements for risk balancing (section 1412(b)(5)(B)), EPA will finalize the LT2ESWTR concurrently with the Stage 2 DBPR to ensure simultaneous protection from microbial and DBP risks.

B. What Is the Regulatory History of the Stage 2 DBPR?

The first rule to regulate DBPs was promulgated on November 29, 1979. The Total Trihalomethanes Rule (44 FR 68624) (USEPA 1979) set an MCL of 0.10 mg/L for total trihalomethanes (TTHMs). Compliance was based on the running annual average (RAA) of quarterly averages of all samples collected throughout the distribution system. This TTHM standard applied only to community water systems using surface water and/or ground water that served at least 10,000 people and added a disinfectant to the drinking water during any part of the treatment process.

Under the Surface Water Treatment Rule (SWTR) (54 FR 27486, June 29, 1989) (USEPA 1989a), EPA set MCLGs of zero for *Giardia lamblia,* viruses, and *Legionella;* and promulgated NPDWRs for all public water systems using surface water sources or ground water sources under the direct influence of surface water. The SWTR includes treatment technique requirements for filtered and unfiltered systems that are intended to protect against the adverse health effects of exposure to *Giardia lamblia,* viruses, and *Legionella,* as well as other pathogenic organisms.

EPA also promulgated the Total Coliform Rule (TCR) on June 29, 1989 (54 FR 27544)(USEPA 1989b) to provide protection from microbial contamination in distribution systems of all types of public water supplies. The TCR established an MCLG of zero for total and fecal coliform bacteria, and an MCL based on the percentage of positive samples collected during a compliance period. Under the TCR, no more than 5 percent of distribution system samples collected in any month may contain coliform bacteria.

Together, the SWTR and the TCR were intended to address risks associated with microbial pathogens that might be found in source waters or associated with distribution systems. However, while reducing exposure to pathogenic organisms, the SWTR also increased the use of disinfectants in some public water systems and, as a result, exposure to DBPs in those systems.

In 1992, prompted by concerns about health risk tradeoffs between disinfection byproducts and microbial pathogens, EPA initiated a negotiated rulemaking with a wide range of stakeholders. The negotiators included representatives of State and local health and regulatory agencies, public water systems, elected officials, consumer groups, and environmental groups. The Regulatory Negotiating Committee met from November 1992 through June 1993. Following months of intensive discussions and technical analyses, the Regulatory Negotiating Committee recommended the development of three sets of rules: an Information Collection Rule, a two-staged approach for regulating DBPs, and an ''interim'' Enhanced Surface Water Treatment Rule (IESWTR) to be followed by a ''final'' Enhanced Surface Water Treatment Rule (USEPA 1996a, USEPA 1998c, USEPA 1998d). EPA took the first step towards implementing this strategy by proposing

the Stage 1 DBPR and IESWTR in 1994. Congress affirmed the phased microbial and disinfection byproduct rulemaking strategy in the 1996 SDWA Amendments by requiring that EPA develop these three sets of rules on a specific schedule that stipulates simultaneous promulgation of requirements governing microbial protection and DBPs.

In March 1997, the Agency established the Microbial and Disinfection Byproduct (M–DBP) Advisory Committee under the Federal Advisory Committee Act (FACA) to collect, share, and analyze new information and data available since the 1994 proposals of the Stage 1 DBPR and the IESWTR, as well as to build consensus on the regulatory implications of the new information. The Advisory Committee consisted of 17 members representing EPA, State and local public health and regulatory agencies, local elected officials, drinking water suppliers, chemical and equipment manufacturers, and public interest groups. The Advisory Committee met five times in March through July 1997 to discuss issues related to the IESWTR and the Stage 1 DBPR. The Advisory Committee reached consensus on a number of major issues that were incorporated into the Stage 1 DBPR and the IESWTR.

The Stage 1 DBPR and IESWTR, finalized in December 1998, were the first rules to be promulgated under the 1996 SDWA Amendments (USEPA 1998c and 1998d). The Stage 1 DBPR applies to all community and nontransient noncommunity water systems that add a chemical disinfectant to water. The rule established maximum residual disinfectant level goals (MRDLGs) and enforceable maximum residual disinfectant level (MRDL) standards for three chemical disinfectants—chlorine, chloramine, and chlorine dioxide; maximum contaminant level goals (MCLGs) for three THMs, two haloacetic acids (HAAs), bromate, and chlorite; and enforceable maximum contaminant level (MCL) standards for TTHM, five haloacetic acids (HAA5), chlorite, and bromate calculated as running annual averages (RAAs). The Stage 1 DBPR uses TTHMs and HAA5 as indicators of the various DBPs that are present in disinfected water. Under the Stage 1 DBPR, water systems that use surface water or ground water under the direct influence of surface water and use conventional filtration treatment are required to remove specified percentages of organic materials, measured as total organic carbon (TOC), that may react with disinfectants to form DBPs. Removal is achieved through enhanced coagulation or enhanced softening, unless a system meets alternative compliance criteria.

EPA finalized the IESWTR at the same time as the Stage 1 DBPR to ensure simultaneous compliance and address risk tradeoff issues. The IESWTR applies to all water systems that use surface water or ground water under the direct influence of surface water that serve at least 10,000 people. The purpose of the IESWTR is to improve control of microbial pathogens in drinking water, specifically the protozoan *Cryptosporidium.*

The Filter Backwash Recycle Rule (FBRR) and the Long Term 1 Enhanced Surface Water Treatment Rule (LT1ESWTR) round out the first group of regulations balancing microbial and DBP risks. EPA promulgated the FBRR in 2001 (USEPA 2001c) and the LT1ESWTR in 2002 (USEPA 2002b) to increase protection of finished drinking water supplies from contamination by *Cryptosporidium* and other microbial pathogens. The LT1ESWTR extends protection against *Cryptosporidium* and other disease-causing microbes to water systems that use surface water or ground water under the direct influence of surface water that serve fewer than 10,000 people. While the Ground Water Rule, proposed in May 2000, (USEPA 2000h) will add significant protection from pathogens in vulnerable ground water systems, it does not pose as many risk-risk tradeoff considerations as the surface water rules because only a small percentage of ground water systems subject to the Stage 2 DBPR have high DBP levels.

EPA reconvened the Advisory Committee in March 1999 to develop recommendations on issues pertaining to the Stage 2 DBPR and LT2ESWTR. The Advisory Committee collected, developed, and evaluated new information that became available after the Stage 1 DBPR was published. The Information Collection Rule provided new data on DBP exposure, and control; it also included new data on occurrence and treatment of pathogens. The unprecedented amount of information collected under the Information Collection Rule was supplemented by a survey conducted by the National Rural Water Association, data provided by various States, the Water Utility Database (which contains data collected by the American Water Works Association), and Information Collection Rule Supplemental Surveys. This large body of data allowed the Advisory Committee to reach new conclusions regarding DBP exposure and new treatment options.

After analyzing the data, the Advisory Committee reached three significant conclusions that led the Advisory Committee to recommending further control of DBPs in public water systems. The data from the Information Collection Rule show that the RAA compliance calculation allows elevated DBP levels to regularly occur at some locations in the system when the overall average at all locations is below the MCL. Customers served at those sampling locations that regularly exceed the MCLs are experiencing higher exposure compared to customers served at locations that consistently meet the MCLs.

Second, the new data demonstrated how single samples can be substantially above the MCLs. The new information showed that it is possible for customers to receive drinking water with concentrations of DBPs up to 75% above the MCLs even when their water system is in compliance with the Stage 1 DBPR. Studies have shown that DBP exposure during short, critical time windows may adversely impact reproductive and developmental health.

Third, data from the Information Collection Rule revealed that the highest TTHM and HAA5 levels are not always located at the maximum residence time monitoring sites specified by the Stage 1 DBPR. These sites were required for monitoring by the Stage 1 DBPR because previous data suggested that water in the distribution system for the maximum residence time would have the highest TTHM levels. The fact that the locations with the highest DBP levels varied in different public water systems indicates that the Stage 1 DBPR monitoring sites may not be representative of the high DBP concentrations that actually exist in distribution systems, and additional monitoring is needed to identify distribution system locations with elevated DBP levels. This information encouraged the Advisory Committee to recommend additional measures to identify locations with high LRAAs. Section IV provides a complete discussion of the new occurrence data.

The analysis of the new data also indicates that certain technologies are effective at reducing DBP concentrations. Bench- and pilot-scale studies for granular activated carbon (GAC) and membrane technologies required by the Information Collection Rule provided information on the effectiveness of the two technologies. Other studies found UV light to be highly effective for inactivating *Cryptosporidium* and *Giardia* at low doses without promoting the formation of DBPs (Malley *et al.* 1996; Zheng *et al.*

1999). This new treatment information added to the treatment options available to utilities for controlling DBPs beyond the requirements of the Stage 1 DBPR.

New data on the health effects of DBPs also influenced the Advisory Committee's recommendation to further regulate DBPs. Although bladder cancer risks were the focus of the Stage 1 M–DBP negotiations, potential reproductive and developmental health effects were central to the Stage 2 M–DBP Advisory Committee discussions. Recent human epidemiology studies and animal toxicology studies have both shown associations between chlorinated drinking water and reproductive and developmental health effects such as spontaneous abortion, stillbirth, neural tube defects, pre-term delivery, intrauterine growth retardation, and low birth weight. A critical review of the epidemiology literature pertaining to reproductive and developmental effects of exposure to DBPs completed in 2000 (Reif *et al.* 2000) concluded that ''the weight of evidence from the epidemiological studies also suggests that they [DBPs] are likely to be reproductive toxicants in humans under appropriate exposure conditions * * * and that measures aimed at reducing the concentrations of byproducts could have a positive impact on public health.''

While there has been substantial research to date, the Advisory Committee recognized that significant uncertainty remains regarding the risk associated with DBPs in drinking water. The Advisory Committee carefully considered the analyses described previously, as well as costs and potential impacts on public water systems, and concluded that a targeted protective public health approach should be taken to address exposure to DBPs beyond the requirements of the Stage 1 DBPR. After reaching this conclusion, the Advisory Committee developed an Agreement in Principle (USEPA 2000g) that laid out their recommendations on how to further control DBPs in public water systems.

In the Agreement in Principle, the Advisory Committee recommended maintaining the MCLs for TTHM and HAA5 at 0.080 mg/L and 0.060 mg/L respectively, but changing the compliance calculation in two phases to facilitate systems moving from the running annual average (RAA) calculation to a locational running annual average (LRAA) calculation. In the first phase, systems would continue to comply with the Stage 1 DBPR MCLs as RAAs and, at the same time, comply with MCLs of 0.120 mg/L for TTHM and 0.100 mg/L for HAA5 calculated as LRAAs. RAA calculations average all samples collected within a distribution system over a one-year period, but LRAA calculations average all samples taken at each individual sampling location in a distribution system during a one-year period. Systems would also carry out an Initial Distribution System Evaluation (IDSE) to select new compliance monitoring sites that more accurately reflect higher TTHM and HAA5 levels occurring in the distribution system. The second phase of compliance would require MCLs of 0.080 mg/L for TTHM and 0.060 mg/L for HAA5 calculated as LRAAs at individual monitoring sites identified through the IDSE.

The Agreement in Principle also provided recommendations for simultaneous compliance with the LT2ESWTR so that the reduction of potential health hazards of DBPs does not compromise microbial protection. The recommendations for the LT2ESWTR included treatment requirements for *Cryptosporidium* based on the results of source water monitoring, a toolbox of options for providing additional treatment at high risk facilities, use of microbial indicators to reduce *Cryptosporidium* monitoring burden on small systems, and future monitoring to determine if source water quality remains constant after completion of initial monitoring. The Agreement also encouraged EPA to develop guidance and criteria to facilitate the use of UV light for compliance with drinking water disinfection requirements. The complete text of the Agreement in Principle (USEPA 2000g) can be found at the edocket Web site *(http://www.epa.gov/edocket).*

After extensive analysis and investigation of available data and rule options considered by the Advisory Committee, EPA is proposing a Stage 2 DBPR control strategy that is consistent with the key elements of the Agreement in Principle signed in September 2000 by the participants in the Stage 2 M–DBP Advisory Committee. EPA determined that the risk-targeting measures recommended in the Agreement in Principle will require only those systems with the greatest risk to make treatment and operational changes and will maintain simultaneous protection from the potential health hazards of DBPs and microbial contaminants. EPA has carefully evaluated and expanded upon the recommendations of the Advisory Committee to more fully develop today's proposal. EPA also made simplifications where possible to minimize complications for public water systems as they transition to compliance with the Stage 2 DBPR while expanding public health protection. The proposed requirements of the Stage 2 DBPR are described in detail in section V of this preamble.

C. How Were Stakeholders Involved in Developing the Stage 2 DBPR?

1. Federal Advisory Committee Process

The Stage 2 M–DBP Advisory Committee consisted of 21 organizational members representing EPA, State and local public health and regulatory agencies, local elected officials, Native American Tribes, large and small drinking water suppliers, chemical and equipment manufacturers, environmental groups, and other stakeholders. Technical support for the Advisory Committee's discussions was provided by a technical working group established by the Advisory Committee. The Advisory Committee held ten meetings to discuss issues pertaining to the Stage 2 DBPR and LT2ESWTR from September 1999 to July 2000 which were open to the public. There was also an opportunity for public comment at each meeting.

In September 2000, the Advisory Committee signed the Agreement in Principle, a full statement of the consensus recommendations of the group. The agreement was published by EPA in a December 29, 2000 **Federal Register** notice (65 FR 83015), together with the list of committee members and their organizations. The Agreement is divided into Parts A and B. The recommendations in each part stand alone and are independent of one another. The entire Advisory Committee reached consensus on Part A, which contains provisions that directly apply to the proposed Stage 2 DBPR and LT2ESWTR. The full Advisory Committee, with the exception of the National Rural Water Association (NRWA), also agreed to Part B, which has recommendations for future activities by EPA in the areas of distribution systems and microbial water quality criteria.

2. Other Outreach Processes

EPA received valuable input from small system operators as part of an Agency outreach initiative under the Regulatory Flexibility Act (RFA). EPA also conducted outreach conference calls to solicit feedback and information from Small Entity Representatives (SERs) on issues related to Stage 2 DBPR impacts on small systems. The Agency consulted with State, local, and Tribal governments on the proposed Stage 2 DBPR. Section VIII includes a complete

description of the many stakeholder activities which contributed to the development of the Stage 2 DBPR.

The Agency held two meetings to discuss consecutive system issues relevant to the proposal (February 22–23, 2001 in Denver, CO and March 28, 2001 in Washington, DC). Representatives from States, EPA Regions, and public water systems participated in the discussions. EPA also briefed the National Drinking Water Advisory Committee at their November 2001 meeting on consecutive system issues associated with the rule to receive input on the implementation strategy selected. This Advisory Committee generally supported EPA's approach. Section V describes EPA's analysis of consecutive system issues, comments and input received during these sessions, and how the proposed requirements will apply to consecutive systems. EPA also consulted with the Science Advisory Board in December 2001 on the requirements of the Stage 2 DBPR.

Finally, EPA posted a pre-proposal draft of the Stage 2 DBPR preamble and regulatory language on an EPA Internet site *(http://www.epa.gov/safewater/mdbp/st2dis.html)* on October 17, 2001. This public review period allowed readers to comment on the Stage 2 DBPR's consistency with the Agreement in Principle of the Stage 2 M–DBP Advisory Committee. EPA received important suggestions on this pre-proposal draft from 14 commenters which included public water systems, State governments, laboratories, and other stakeholders. While EPA will not formally respond to these comments, EPA has carefully considered them in developing today's proposal.

III. Public Health Risk

Chlorine has been widely used as a chemical disinfectant, serving as a principal barrier to microbial contaminants in drinking water. However, the microbial risk reduction attributes of chlorination have been increasingly scrutinized due to concerns about potential increased health risks from exposure to disinfection byproducts, which are formed when certain disinfectants interact with organic and inorganic material in source waters. Since the discovery of chlorination byproducts in drinking water in 1974, numerous toxicological studies have shown several DBPs (*e.g.*, bromodichloromethane, bromoform, chloroform, dichloroacetic acid, trichloroacetic acid and bromate) to be carcinogenic in laboratory animals. These findings of carcinogenicity influenced EPA to promulgate the TTHM Rule in 1979 and the Stage 1 DBPR in 1998. The Stage 1 DBPR primarily addressed possible carcinogenic effects (*e.g.*, bladder, colon and rectal cancers) reported in both human epidemiology and laboratory animal studies. Since the Stage 1 DBPR, new health studies continue to support an association between bladder, colon and rectal cancers from long-term exposure to chlorinated surface water. In addition to cancer effects, recent studies have reported associations between use of chlorinated drinking water and a number of reproductive and developmental endpoints including spontaneous abortion, still birth, neural tube defect, pre-term delivery, low birth weight and intrauterine growth retardation (small for gestational age). Short-term, high-dose animal screening studies on individual byproducts (*e.g.*, bromodichloromethane (BDCM), and certain haloacetic acids) have also reported adverse reproductive and developmental effects (*e.g.*, whole litter resorption, reduced fetal body weight) that are similar to those reported in the human epidemiology studies. This section discusses the new studies that have become available since promulgation of the Stage 1 DBPR and how they contribute to the weight of evidence for an association between health effects and exposure to chlorinated surface water.

While the Stage 1 DBPR was targeted primarily at reducing long-term exposures to elevated levels of DBPs to address chronic health risks from cancer, the Stage 2 DBPR targets reducing short-term exposures to address potential reproductive and developmental health risks and cancer risks.

Based on the weight of evidence from both the human epidemiology and animal toxicology data on cancer and reproductive and developmental health effects and consideration of the large number of people exposed to chlorinated byproducts in drinking water (approximately 254 million), EPA concludes that: (1) Current reproductive and developmental health effects data support a hazard concern, (2) new cancer data strengthens the evidence of an association of chlorinated water with bladder cancer and suggests an association for colon and rectal cancers, and (3) the combined health data warrant regulatory action beyond the Stage 1 DBPR.

A. Reproductive and Developmental Epidemiology

The following section briefly discusses reproductive and developmental epidemiology information EPA analyzed, some conclusions of these studies and reports, and implications for the Stage 2 DBPR. Further discussion of the implications and EPA's conclusions can be found in the Stage 2 Economic Analysis (USEPA 2003i).

EPA has evaluated recently published epidemiological studies examining the relationship between exposure to contaminants in chlorinated surface water and adverse reproductive and developmental outcomes. EPA also considered critical reviews of the epidemiological literature by Reif *et al.* (2000), Bove *et al.* (2002), and Nieuwenhuijsen *et al.* (2000). Based on these evaluations, EPA believes that the reproductive and developmental epidemiology data contribute to the weight of evidence on the potential health risks from exposure to chlorinated drinking water. Although the data are not suitable for a quantitative risk assessment at this time, due in part to inconsistencies in the findings, they do suggest that exposure to DBPs is a potential reproductive and developmental health hazard.

1. Reif *et al.* 2000

Reif *et al.* (2000) completed a critical review of the epidemiology literature pertaining to reproductive and developmental effects of exposure to disinfection byproducts in drinking water as a report to Health Canada. The review focused on 16 peer-reviewed scientific manuscripts and published reports and evaluated associations between DBP exposure and outcomes grouped as effects on: (1) Fetal growth—low birth weight (<2500g); very low birth weight (<1500g); preterm delivery (<37 weeks of gestation) and intrauterine growth retardation (or small for gestational age); (2) fetal viability (spontaneous abortion and stillbirth) and (3) fetal malformations (all malformations, oral cleft defects, major cardiac defects, neural tube defects, and chromosomal abnormalities).

a. *Fetal growth.* Reif *et al.* (2000) found inconsistent epidemiological evidence for an association between DBPs and fetal growth. Some studies found weak but statistically significant associations (Gallagher *et al.* 1998; Bove *et al.* 1992 and 1995), while two studies found no association (Dodds *et al.* 1999; and Savitz *et al.* 1995) with fetal growth.

b. *Fetal viability.* Reif *et al.* 2000's review of the literature found inconsistencies in the epidemiological evidence for the association between DBP exposure and fetal viability. For instance, the study by Waller *et al.* 1998 found an apparent dose-dependent increase in rates of spontaneous

abortions associated with TTHMs in California. On the other hand, Savitz *et al.* (1995) found little evidence of an association using either the concentration of TTHM ≥81 µg/L or a dose estimate based on the amount of tap water consumed. An increased risk of stillbirth was reported for women in Nova Scotia by Dodds *et al.* 1999, but in New Jersey, Bove *et al.* (1992, 1995) found little evidence of an association with TTHM at 80 µg/L, but did report a weak association between stillbirth and use of surface water systems. Aschengrau *et al.* (1993) found an association between stillbirth and the use of a chlorinated vs. chloraminated surface water supply, but not for exposure to surface water.

c. *Fetal malformations and other developmental anomalies.* Reif *et al.* (2000) considered the data for congenital anomalies to be inconsistent across the six studies that have explored these outcomes. For example, two of the four studies on neural tube defects (Bove *et al.* 1995; Magnus *et al.* 1999) reported significant excess risks, but the remaining two studies (Dodds *et al.* 1999; Klotz and Pyrch *et al.* 1999) did not. These studies found lower risks or no evidence of an association with TTHM. However, those studies were conducted in locations with either very low or high concentrations of DBPs which may have limited the contrast in exposures, thereby reducing the ability to detect increased risks. An assessment of congenital anomalies is also difficult due to the relatively small number of cases available for evaluation.

Overall, Reif *et al.* (2000) conclude that the weight of evidence from the epidemiological studies suggest that ''DBPs are likely to be reproductive toxicants in humans under appropriate exposure conditions.'' Reif *et al.* comment that data from animal studies of individual DBPs provide biological plausibility for the effects observed in epidemiological studies. Although the authors recognize that the ''data are primarily at the stage of hazard identification,'' they conclude that ''measures aimed at reducing the concentrations of byproducts could have a positive impact on public health.''

2. Bove *et al.* 2002

Bove *et al.* (2002) conducted a qualitative review of 14 epidemiological studies that evaluated possible developmental and reproductive endpoints associated with exposure to chlorination byproducts in drinking water. Similar to Reif *et al.*, Bove *et al.* evaluated associations between DBP exposure and outcomes grouped as effects on (1) fetal growth—small for gestational age (SGA) as defined in each study (usually defined as the fifth or tenth percentile weight by gestational week of birth); (2) fetal viability—spontaneous abortion and stillbirth; and (3) fetal malformations (neural tube defects, oral clefts, and cardiac defects).

a. *Fetal growth.* Bove *et al.* found that, although the studies that evaluated SGA had several limitations, three studies out of eight (Kramer *et al.* 1992, Bove *et al.* 1995, and Gallagher *et al.* 1998) ''provided moderate evidence for a causal relationship between a narrow definition of SGA * * * and TTHM levels that could be found currently in some U.S. public water systems.'' They also concluded that the study with the best exposure assessment found the strongest association between SGA and TTHM exposure (Gallagher *et al.* 1998). One study found a very weak association (Dodds *et al.* 1999) and the other four did not observe an association (Yang *et al.* 2000, Kanitz *et al.* 1996, Kallen *et al.* 2000, and Jaakkola *et al.* 2001).

b. *Fetal viability.* Bove *et al.* evaluated three studies on spontaneous abortion and three studies on stillbirth. Again, Bove *et al.* found that the study employing the best methods found the strongest association between TTHM exposure and spontaneous abortions (Waller *et al.* 1998). The other two studies (Savitz *et al.* 1995 and Aschengrau *et al.* 1989) found weak associations. Two of the studies investigating stillbirths found an association between stillbirths and chlorinated surface water (Dodds *et al.* 2001 and Aschengrau *et al.* 1993). The third study (Bove *et al.* 1995) found no association, however this study did not evaluate individual THM levels or cause of death information.

c. *Fetal malformations.* Bove *et al.* evaluated seven studies that investigated the relationship between birth defects and DBP exposure. This evaluation found ''consistency among these studies in the findings for neural tube defects and oral cleft defects, but not for cardiac defects. Associations were found for neural tube defects in all three studies that examined neural tube defects. These studies also evaluated levels of THM exposure (Bove *et al.* 1995; Dodds *et al.* 1999; Klotz *et al.* 1999).'' Two studies evaluated oral cleft defects and levels of THMs; one found an association with TTHM (Bove *et al.* 1995) and the other found an association with chloroform (Dodds *et al.* 2001). A third study that did not evaluate THM levels did not identify an association with oral cleft defects (Jaakkola *et al.* 2001). Bove *et al.* 1995 found an association between cardiac defects and TTHM, but Dodds *et al.* 1999, 2001 and Shaw *et al.* 1991 did not. An association between chlorination and urinary tract defects was found in the three studies that evaluated that endpoint (Källén *et al.* 2000; Magnus *et al.* 1999; Aschengrau *et al.* 1993).

Bove *et al.* (2002) concluded that the current reproductive and developmental epidemiological database for exposure to chlorinated byproducts in drinking water presents moderate evidence for associations between DBP exposure and SGA, neural tube defects and spontaneous abortion. The authors acknowledged the difficulties in assessing exposure with any precision in the studies reviewed, but held the opinion that misclassification of exposure would tend to underestimate rather than overestimate the risk.

3. Nieuwenhuijsen *et al.* 2000

Nieuwenhuijsen *et al.* (2000) reviewed the toxicological and epidemiological literature and evaluated the potential risk of chlorination DBPs on human reproductive health. The authors state that ''some studies have shown associations for DBPs and other outcomes such as spontaneous abortions, stillbirths and birth defects, and although the evidence for these associations is weaker it is gaining weight.'' Nieuwenhuijsen *et al.* also concluded that, ''although studies report small risks that are difficult to interpret, the large number of people exposed to chlorinated water supplies constitutes a public health concern.''

4. Additional Epidemiology Studies

Three new reproductive and developmental epidemiological studies were completed that were not included in the Reif *et al.* 2000, Bove *et al.* 2002, or Nieuwenhuijsen *et al.* 2000 literature reviews.

Waller *et al.* 2001, recalculated the total trihalomethane exposures from their original publication (Waller *et al.* 1998) to evaluate two exposure assessment methods (closest site and utility-wide average). The new calculations were intended to reduce exposure misclassification by employing weighting factors and subset analyses. As in the 1998 publication, the new methods found a relationship between spontaneous abortion and THM exposure, although the unweighted utility-wide point estimate was lower than reported in the original manuscript.

Hwang *et al.* 2002, assessed the effect of water chlorination byproducts on specific birth defects in Norway by

classifying exposure on the basis of chlorination (yes/no) and amount of natural organic matter in the water. Statistically significant associations with exposure were found for risks of any birth defect, cardiac, respiratory, and urinary tract defects. For specific birth defects, a statistically significant association was found for a defect of the septum in the heart.

Windham *et al.*, 2003, assessed the relationship between exposure to THMs in drinking water and characteristics of the menstrual cycle among 403 women who provided daily urine samples for an average of 5.6 cycles. Women whose tap water had TTHM levels more than 0.060 mg/l had statistically significantly shorter menstrual cycles than women whose tap water had lower TTHMs. On average, the menstrual cycles of women with the higher levels of TTHMs were one day shorter than cycles of women with the lower levels (adjusted difference: −1.1 days, 95% confidence interval: −1.8 days to −0.4 days). This shortening occurred during the first half of the cycle, before ovulation (adjusted difference: −0.9 days; 95% confidence interval: −1.6 days to −0.2 days). There were no changes in bleed length or in the regularity of the cycles. Based on their study, Windham *et al.*, 2003, suggested that THM exposure may affect ovarian function, but since this is the first study to examine human menstrual cycle variation in relation to THM exposure, more research is needed to confirm the relationship. The public health implication of a small reduction in menstrual cycle length is not clear, but if THMs are related to disturbances in ovarian function, that might provide insight into the observed associations between THMs and a variety of adverse reproductive outcomes.

EPA's epidemiology research program continues to examine the relationship between exposure to DBPs and adverse developmental and reproductive effects. The Agency is supporting several studies using improved study designs to provide better information for characterizing potential risks. Details on EPA's epidemiology research program can be found at *http://cfint.rtpnc.epa.gov/dwportal/cfm/dwMDBP.cfm*.

B. Reproductive and Developmental Toxicology

Several new reproductive and developmental toxicology studies have become available since the December 1998 Stage 1 DBPR. This discussion presents some conclusions derived from these studies and reports, including hazard identification, as well as implications for the Stage 2 DBPR.

EPA conducted a literature search of animal toxicology studies on chronic and subchronic DBP exposures associated with reproductive and developmental health effects, evaluated the current reproductive and developmental toxicological database for several individual DBPs, and assessed two independent reviews (Tyl 2000 and WHO 2000). As a result of these analyses, EPA has concluded that although the database is not strong enough to quantify risk, it is sufficient to support a hazard concern. This hazard concern supports the need to address potential reproductive and developmental health effects in the Stage 2 DBPR. The following section describes how this conclusion was reached.

1. EPA Analysis and Research

Since the Stage 1 DBPR, EPA has continued to support reproductive and developmental toxicological research on various disinfection byproducts through extramural and intramural research programs. Information on EPA's toxicology programs can be found at *http://www.epa.gov/nheerl/*. These studies, along with data on several DBPs published after the 1998 Stage 1 DBPR, are summarized in the updated children's health document, ''Health Risks to Fetuses, Infants, and Children: A Review'' (USEPA 2003a).

In addition to this compilation of data, EPA has also prepared individual health criteria documents that provide detailed summaries of the relevant new information, as well as an overall characterization of the human health risks from exposure to certain DBPs (USEPA 2003b-USEPA 2003h, USEPA 2003l). From these new evaluations, EPA has concluded that several new studies on individual byproducts contribute to the weight of evidence for an association between DBP exposure and adverse effects on the developing fetus and reproduction. These effects include fetal loss, cardiovascular effects, and male reproductive effects and are associated with bromodichloromethane (BDCM), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), bromochloroacetic acid (BCAA), and dibromoacetic acid (DBAA). The data from these new studies do not change the MCLGs that were established as a part of the Stage 1 DBPR.

2. Tyl 2000

Tyl (2000) conducted a comprehensive review of the reproductive and developmental toxicology literature on DBPs representing over thirty-five studies. Adverse effects reported by these studies include developmental effects, whole litter resorption, reduced fetal body weights, and male reproductive effects (*e.g.*, inhibited spermiation, increased abnormal sperm). Many of these studies are categorized as high-dose, short-term screening studies that can be used to assess potential hazard (Table III–1), while the long term, two-generation reproduction studies could be an appropriate basis for quantitative risk assessment.

Disinfectant/DBP	Screening [1]	Developmental [2]	Two-generation [3] reproductive
Chlorine		✔	
Chlorine Dioxide	✔	✔	
Chloramine		✔	
Chloroform	✔	✔	✔
Bromoform	✔	✔	✔
Bromodichloromethane	✔	✔	in progress
Dibromochloromethane	✔	✔	
Monochloroacetic acid	✔	✔	
Dichloroacetic acid	✔	✔	
Trichloroacetic acid	✔	✔	
Monobromoacetic acid	✔	✔	
Dibromoacetic acid	✔	✔	in progress
Tribromoacetic acid	✔		
Bromochloroacetic acid	✔		in planning stage
Bromodichloroacetic acid	✔		
Dibromochloroacetic acid	✔		

Disinfectant/DBP	Screening [1]	Developmental [2]	Two-generation [3] reproductive
Chloroacetonitrile	✔		
Dichloroacetonitrile	✔	✔	
Trichloroacetonitrile	✔	✔	
Bromoacetonitrile	✔	✔	
Dibromoacetonitrile	✔		
Tribromoacetonitrile			
Bromochloroacetonitrile	✔	✔	
Propanal	✔	✔	
1,1 Dichloropropanone	✔		
Hexachloropropanone	✔		
Dichloromethane	✔		
MX	✔	✔	
Bromate	✔		
Chlorite	✔	✔	✔

✔ denotes the availability of at least one study in the following categories.
[1] Screening studies are for hazard identification. These types of studies include the following: whole embryo culture, NTP 35-day screening studies, Chernoff-Kavlock and its modified version, and short-term male reproductive toxicity screen.
[2] Developmental studies are used for dose-response determinations.
[3] Two-generation reproductive studies are multi-generation reproductive toxicity studies used for dose-response determinations.

Tyl concluded that, ''The screening studies, performed for a number of DBPs, are 'adequate' and 'sufficient' only to detect potent reproductive/developmental toxicants for hazard identification.'' Tyl further confirms that the database identifies certain DBPs with potential reproductive or developmental effects (Table III–2) and these are discussed further in the next section.

TABLE III–2.—POTENTIAL HAZARDS OF DBPS FOR REPRODUCTIVE AND DEVELOPMENTAL EFFECTS (ADAPTED FROM TYL, 2000)

Type of hazard	Disinfection byproducts
Developmental defects	TCAA, DCAA, MCAA and chlorite.
Whole litter resorption	Chloroform, bromoform, BDCM, DBCM, DCAA, TCAA, DCAN, and TCAN.
Fetotoxicity (reduced fetal body weights, increased variations)	Chloroform, BDCM, DBCM, DCAA, TCAA, DCAN, TCAN, DBAN, BCAN, MCAN.
Male reproductive effects (spermatotoxic)	DCAA, DBAA, BDCM.

a. *Developmental defects.* Tyl noted that adverse developmental effects that were reported from whole embryo culture tests on the developing heart, neural tube, eye, pharyngeal arch, and somites tended to be associated with haloacetic acids tested at high doses (Hunter *et al.* 1996; Saillenfait *et al.* 1995, Smith *et al.* 1989). Cardiovascular effects were also observed in vivo for TCAA and DCAA from developmental segment II toxicity studies at high doses (Smith *et al.* 1988, 1990).

b. *Whole litter resorption.* Whole litter resorption, likened to miscarriage or spontaneous abortion by Tyl 2000, was also observed at high doses in vivo for a range of DBPs as indicated in Table III–2 (Murray *et al.* 1979, Balster and Borzellca, 1982, Narotsky *et al.* 1992; 1997 a, b; Bielmeier *et al.* 2001; Smith *et al.* 1990; Smith *et al.* 1988). Tyl noted that similar effects were observed in several epidemiology studies.

c. *Fetal toxicity.* Fetal toxic effects such as reduced fetal body weights and increased variation were observed at high doses in vivo for a range of DBPs (*e.g.*, chloroform, BDCM, DBCM, DCAA, TCAA, DCAN, TCAN, DBAN, BCAN) (Thompson *et al.* 1974; Schwetz *et al.* 1974; Murray *et al.* 1979; Ruddick *et al.* 1983; Narotsky *et al.* 1992, Balster and Borzelleca, 1982; Smith *et al.* 1990). Again, Tyl noted a similarity in effects observed in epidemiology studies.

d. *Male reproductive effects.* Animal toxicology studies report increased risks of adverse effects on the male reproductive system from high doses of haloacetic acids and other DBPs that have not been studied in human epidemiology studies. Male reproductive effects (*e.g.*, inhibited spermiation, reduced epididymus, sperm number and motility, increased abnormal sperm, testicular damage and inhibited in vitro fertilization) were reported for DCAA, DBAA, TCAA and BDCM (Toth *et al.* 1992, Linder *et al.* 1997a, b; Linder *et al.* 1994a, b; Cosby and Dukelow 1992). Dr. Tyl noted that the adverse effects observed in the male reproductive toxicity screening studies (Toth *et al.* 1992; Linder *et al.* 1994a, b; 1997a, b) are confounded by a short dosing regimen and administration of test doses to only adult males.

From her review of the comprehensive animal toxicology database on reproductive and developmental health effects from DBP exposure, Dr. Tyl concludes that ''some DBPs have an intrinsic capacity to do harm, specifically to the developing conceptus and the male (and possibly the female) reproductive system''. She concludes that ''there is hazard to development from the haloacetic acids (TCAA, DCAA, MCAA) and acetate; to development from chloroform, bromoform, BDCM, DBCM, DCAA, TCAA, DCAN, and TCAN expressed as full litter resorption (which most likely indicates maternal endocrine/uterine effects); and fetotoxicity for chloroform, BDCM, DBCM, DCAA, TCAA, DCAN, TCAN, DBAN, BCAN, CAN, acetaldehyde, and possibly formaldehyde. Reproductive hazard exists for DCAA, DBAA, and possibly formaldehyde in males and for TCE and possibly formaldehyde in females.''

3. World Health Organization Review of the Reproductive and Developmental Toxicology Literature (2000)

The International Programme on Chemical Safety (IPCS) published an evaluation of Disinfectants and DBPs in its *Environmental Health Criteria* monograph series (WHO 2000). In this review of the toxicology data on reproductive and developmental effects from DBP exposure, the World Health Organization (WHO) concludes that although the data on these effects are not as robust as the cancer database, these effects are of potential health concern. The WHO concludes that reproductive effects in females have been principally embryolethality and fetal resorptions associated with the haloacetonitriles (trichloroacetonitrile, dichloroacetonitrile, bromochloroacetonitrile, and dibromoacetonitrile) and the dihaloacetates, while DCAA and DBAA have both been associated with adverse effects on male reproduction.

4. New Studies

Christian *et al.* (2001) conducted a developmental toxicity study with pregnant New Zealand White rabbits exposed to BDCM in drinking water at concentrations of 0, 15, 150, 450, and 900 ppm in drinking water on gestation days 6–29. The no observed adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL) identified for maternal toxicity in this study were 13.4 mg/kg-day (150 ppm) and 35.6 mg/kg-day (450 ppm), respectively, based on decreased body weight gain. The developmental NOAEL was 55.3 mg/kg-day (900 ppm) based on absence of statistically significant, dose-related effects at any tested concentration. Christian *et al.* (2001) also conducted a developmental study of BDCM in a second species, Sprague-Dawley rats. Rats were exposed to BDCM in the drinking water at concentrations of 0, 50, 150, 450, and 900 ppm on gestation days 6 to 21. The concentration-based maternal NOAEL and LOAEL for this study were 150 ppm and 450 ppm, respectively, based on statistically significant, persistent reductions in maternal body weight and body weight gains. Based on the mean consumed dosage of bromodichloromethane, these concentrations correspond to doses of 18.4 mg/kg-day and 45.0 mg/kg-day, respectively. The concentration-based developmental NOAEL and LOAEL were 450 ppm and 900 ppm, respectively, based on a significantly decreased number of ossification sites per fetus for the forelimb phalanges (bones of the hand or the foot) and the hindlimb metatarsals and phalanges. These concentrations correspond to mean consumed doses of 45.0 mg/kg-day and 82.0 mg/kg-day, respectively.

Christian *et al.* (2002b) summarized the results of a two-generation reproductive toxicity study on bromodichloromethane conducted in Sprague-Dawley rats. Bromodichloromethane was continuously provided to test animals in the drinking water at concentrations of 0, 50, 150, or 450 ppm. Average daily doses estimated for the 50, 150, and 450 ppm concentrations were reportedly 4.1 to 12.6, 11.6 to 40.2, and 29.5 to 109 mg/kg-day, respectively. The parental NOAEL and LOAEL were 50 and 150 ppm, respectively, based on statistically significant reduced body weight and body weight gain; F1 and F2 generation pup body weights were reduced in the 150 and 450 ppm groups during the lactation period after the pups began to drink the water provided to the dams. Body weight and body weight gain were also reduced in the 150 and 450 ppm F1 generation males and females. A marginal effect on estrous cyclicity was observed in F1 females in the 450 ppm exposure group. Small (≤6%), but statistically significant, delays in F1 generation sexual maturation occurred at 150 (males) and 450 ppm (males and females) as determined by timing of vaginal patency or preputial separation. The study's authors considered these effects to be a secondary response associated with reduced body weight, which appears to be dehydration brought about by taste aversion to the compound. The results of this study identify NOAEL and LOAEL values for reproductive effects of 50 ppm (4.1 to 12.6 mg/kg-day) and 150 ppm (11.6 to 40.2 mg/kg-day), respectively, based on delayed sexual maturation.

Bielmeier *et al.* (2001) conducted a series of experiments to investigate the mode of action in bromodichloromethane-induced full litter resorption (FLR). The study included a strain comparison of F344 and Sprague-Dawley (SD) rats. In the strain comparison experiment, female SD rats (13 to 14/dose group) were dosed with 0, 75, or 100 mg/kg-day by aqueous gavage in 10% Emulphor® on GD 6 to 10. F344 rats (12 to 14/dose group) were dosed with 0 or 75 mg/kg-day administered in the same vehicle. The incidence of FLR in the bromodichloromethane-treated F344 rats was 62%, while the incidence of FLR in SD rats treated with 75 or 100 mg/kg-day of bromodichloromethane was 0%. Both strains of rats showed similar signs of maternal toxicity, and the percent body weight loss after the first day of dosing was comparable for SD rats and the F344 rats that resorbed their litters. The rats were allowed to deliver and pups were examined on postnatal days 1 and 6. Surviving litters appeared normal and no effect on post-natal survival, litter size, or pup weight was observed. The series of experiments conducted by Bielmeier *et al.* (2001) identified a LOAEL of 75 mg/kg-day (the lowest dose tested) based on FLR in F344 rats. A NOAEL was not identified. Mechanistic studies indicate that BDCM-induced pregnancy loss is likely to be luteinizing hormone (LH)-mediated (Bielmeier *et al.*, 2001). It is possible that BDCM alters LH levels by disrupting the hypothalamic-pituitary-gonadal axis or by altering the responsiveness of the corpora lutea to LH. Since these possible mechanisms are potentially relevant to pregnancy maintenance in humans, EPA believes the finding of BDCM-induced pregnancy loss in F344 rats is relevant to risk assessment, and may provide insight into the epidemiological finding of increased risk of spontaneous abortion associated with consumption of BDCM (Waller *et al.* 1998, 2001).

Christian *et al.* (2002a) recently completed a two-generation drinking water study of DBA in rats. Male and female Sprague-Dawley rats (30/sex/exposure group) were administered DBA in drinking water at concentrations of 0, 50, 250, or 650 ppm continuously from initiation of exposure of the parental (P) generation male and female rats through weaning of the F_2 offspring. Based on testicular histomorphology indicative of abnormal spermatogenesis in P and F_1 males, the parental and reproductive/developmental toxicity LOAEL and NOAEL are 250 and 50 ppm, respectively.

Previous studies by EPA have reported adverse effects of DBA, administered via oral gavage, on spermatogenesis that impacted male fertility (Linder *et al.* 1994a, 1995, 1997a) at doses-comparable to those achieved in the Christian *et al.* (2002a) study. Based on these studies collectively, it is clear that DBA is spermatotoxic. Moreover, Veeramachaneni *et al.* (2000) reported in an abstract that sperm from male rabbits exposed to DBA *in utero* from gestation days 15 and throughout life reduced the fertility of artificially inseminated females as evidenced by reduced conceptions. When published, this study may support the evidence that DBA is a male reproductive system toxicant .

In addition, research on DBA by Klinefelter *et al.* (2001) has

demonstrated statistically significant delays in both vaginal opening and preputial separation using the body weight on the day of acquisition (postnatal day 45) as the co-variant. This was not found by Christian *et al* (2002a) using the body weight at weaning as the statistical covariant. However, the authors analyzed the data for preputial separation and vaginal opening with body weight on the day of weaning as a co-variant rather than body weight on the day of acquisition, *i.e.*, the day that the prepuce separates or the day the vagina opens. It is likely that there was an increase in body weight from postnatal day 21 (weaning) until preputial separation (day 45) that was independent of the delay in sexual maturation.

Although the Christian *et al.* (2002a) study was conducted in accordance with EPA's 1998 testing guidelines, EPA has incorporated newer, more sophisticated measures into recent intramural and extramural studies that have not yet been incorporated into the testing guidelines. Such measures include measuring changes in specific proteins in the sperm membrane proteome and fertility assessments via in utero insemination. EPA believes that additional research is needed, utilizing these newer toxicological measures, to clarify the extent to which DBA poses human reproductive or developmental risk. The database on male reproductive effects from exposure to DBA is incomplete and is not suitable for quantitative risk assessment at this time. It does, however, identify reproductive effects as an area of concern.

C. Conclusions Drawn From the Reproductive and Developmental Health Effects Data

EPA believes that the weight of evidence of the best available science, in conjunction with the widespread exposure, supports regulatory changes that target peak DBP exposures specifically through the Stage 2 DBPR. Several epidemiology studies found statistically significant associations between exposure to chlorinated drinking water and fetal growth, spontaneous abortion, stillbirth, and neural tube defects. Although uncertainties remain and the current database does not support a quantitative reproductive and developmental risk assessment for most of the DBPs, the weight of evidence provides an indication of a hazard concern that warrants additional regulatory action beyond the Stage 1 DBPR.

Biological plausibility for the effects observed in epidemiological studies has been demonstrated through various toxicological studies. Tyl 2000 states that ''effects observed in animal studies included embryonic heart and neural tube defects from haloacetic acids in vitro and in vivo, and full litter resorption, reduced numbers of implants per litter, and reduced fetal body weight per litter were also observed from exposure to specific trihalomethanes. Comparable effects were also observed in children in some (but not all) epidemiological studies, with exposure to trihalomethanes (THMs) usually used as a surrogate for specific DBP classes or individual DBPs, as follows: increased incidences of cardiac defects (Bove *et al.* 1995) and of neural tube defects in children (Bove *et al.* 1995; Dodds *et al.* 1999; Klotz and Pyrch 1998) were reported. Intrauterine growth retardation (IUGR, approximately equivalent to reduced fetal body weights per litter) was reported to be associated with waterborne chloroform (Kramer *et al.* 1992; Bove *et al.* 1995; Gallagher *et al.* 1998). Miscarriage or spontaneous abortion, or stillbirth (approximately equivalent to whole litter resorption, reduced numbers of total and/or live implants per litter, and increased resorptions per litter) were observed by Waller *et al.*, 1998; Dodds *et al.*, 1999; and Bove *et al.*, 1995.''

Similarity of effects between animals and humans lends credence to and strengthens the weight of evidence for an association between adverse reproductive and developmental health effects and exposure to chlorinated surface water. EPA believes that the weight of evidence of both the reproductive and developmental toxicological and epidemiological databases suggests that exposure to DBPs may induce potential adverse health effects on reproduction and fetal development at some DBP exposures. However, additional toxicological work is necessary to identify the mode of action for the effects observed.

D. Cancer Epidemiology

Epidemiological studies on cancer provide valuable information that contributes to the overall evidence on the potential human health hazards from exposure to chlorinated drinking water. In the area of epidemiology, a number of studies have been conducted to investigate the relationship between exposure to chlorinated surface water and cancer. While EPA cannot conclude there is a causal link between exposure to chlorinated surface water and cancer, some studies have found an association between bladder, rectal and colon cancer and exposure to chlorinated surface water.

1. Population Attributable Risk Analysis

Some epidemiological studies have linked the consumption of chlorinated surface waters to an increased risk of two major causes of human mortality in the United States, colorectal and bladder cancers (Cantor 1998). Bladder cancer was chosen as the primary endpoint of concern in the Stage 1 DBPR (USEPA 1998f) economic analysis because it had the most consistent database for a possible association to chlorinated surface water exposure. More studies have considered bladder cancer than any other cancer. EPA used the published mean risk estimates from five studies to quantify the potential range of risk for bladder cancer from DBP exposure. These risks were expressed as a range of population attributable risks (PAR) of 2–17% (USEPA 1998f). This means that if the associations reported in the studies turn out to reflect a causal link, between 2 and 17% of new bladder cancer cases could be attributable to DBPs. This PAR range also represents that portion of the bladder cancer cases that would not have occurred if the exposure to chlorinated drinking water were absent. A complete discussion of the Stage 1 DBPR bladder cancer PAR evaluation, including uncertainties and assumptions, can be found in the Stage 2 DBPR Economic Analysis (USEPA 2003i).

While EPA recognized the limitations of the epidemiological database for making risk estimates, the Agency believed that it was useful for developing an estimate of bladder cancer risk. The PARs were derived from measured risks (Odds Ratios and Relative Risk) based on the number of years exposed to chlorinated surface water. The uncertainties associated with these PAR estimates are largely due to the common prevalence of both the disease (bladder cancer) and exposure (chlorinated drinking water). EPA recognizes that risks from chlorinated drinking water may be lower or higher than those estimated from the epidemiological literature, and that the PAR range could include zero or be higher than 17%.

Using the PARs of 2% and 17%, EPA estimated that the number of possible bladder cancer cases per year potentially associated with exposures to DBPs in chlorinated drinking water could range from 1,100 to 9,300 cases. This was based on the estimate of 54,500 new bladder cancer cases per year nationally, as projected by the National Cancer Institute for 1997. A thorough discussion of cancer studies published prior to 1998 and possible

associations with DBP exposure can be found in the Stage 1 DBPR (USEPA 1998c).

2. New Epidemiological Cancer Studies

New studies published since the Stage 1 DBPR continue to support an association between bladder, colon and rectal cancers and exposure to chlorinated surface water (Yang *et al.* 1998; Koivusalo *et al.* 1998; King *et al.* 2000b). Based on the weight of evidence provided by the cancer epidemiology database, EPA has chosen to use the same PAR analysis to estimate the primary benefits from bladder cancer cases potentially avoided as a consequence of reducing the DBP levels from the Stage 2 DBPR (*see* section VII). For the Stage 2 DBPR analysis, EPA updated the 1997 estimate of new bladder cancer cases per year nationally from 54,500 to 56,500 (projected by the American Cancer Society, 2002) and accounted for the reductions in DBP exposure that were projected for the Stage 1 DBPR.

a. *New bladder cancer studies.* Bladder cancer and chlorinated DBP exposure has historically been the most strongly supported association of all the possible cancers, based on human evidence. Two new studies (Yang *et al.* 1998 and Koivusalo *et al.* 1998) also suggest an association of DBP exposure with bladder cancer. Yang *et al.* 1998 found a positive association between consumption of chlorinated drinking water and bladder cancer. Koivusalo *et al.* (1998) found evidence of increased risk as a function of increasing DBP exposure duration. Long exposure durations (≥45 years for Koivusalo *et al.* 1998) were associated with about a two-fold increase in risk. The new bladder cancer studies continue to support an association and potential for a causal relationship between exposure to chlorination byproducts and risk for bladder cancer.

A new publication by C.M. Villanueva *et al.* (Villanueva *et al.* 2003) reports on their meta-analysis of case-control and cohort studies. This meta-analysis may be useful for improving the estimate of national population attributable risk (fraction of bladder cancer cases in the U.S. that may be attributed to chlorinated drinking water). Compared to EPA's current approach (*i.e.*, providing a range of population attributable risks (PAR)), use of the meta-estimate would provide a more stable result because:

- It provides a single (meta) estimate of the odds ratio from which to calculate the PAR, thereby summarizing the results across studies, thus reducing the influence of geographic and temporal differences.
- It uses three additional high-quality studies not included in the PAR range analysis conducted by EPA (*i.e.*, studies by Koivusalo *et al.* 1998, Doyle *et al.* 1997, and Vena *et al.* 1993).
- It weights the individual studies according to their precision, so more precise estimates (due principally to greater numbers of cases) carry greater statistical weight and therefore have greater influence on the meta-estimate.
- In addition to the primary analysis, the authors conducted an evaluation of the robustness of their conclusions. They examined the sensitivity of estimates to decisions made with respect to exposure definitions, cut points defining exposure groups, inclusion/exclusion of individual studies, and potential publication bias.

The meta-analysis provided at least two meta-estimates that may be useful for estimating national population attributable risk:

- A combined odds ratio for ever-exposure, with confidence intervals and
- A combined dose-response regression slope coefficient, relating increasing odds ratios to additional years of chlorinated drinking water consumption.

EPA conducted an estimate of the impact of using the meta-analysis to provide a perspective on the national population attributable risk. This estimate is based on the author's correction of a minor transcription error in their published manuscript (the appropriate estimate for the King study yields corrected over-all odds ratio for ever-consumers of 1.2 with 95% confidence interval of 1.091 to 1.320, personal communication from M. Kogevinas to M. Messner, 5/19/2003). Assuming 70% of the U.S. population is in the ever-consumed category (based on the chlorinated surface water exposed population), a point estimate of the population attributable risk using the odds ratio from the meta-analysis is 12% (95% interval 6% to 18%). Although EPA's population attributable risk range (2% to 17%) was not intended to convey a quantified level of confidence, it is not vastly different from the meta-analysis' 95% confidence range of 6% to 18%. EPA regards the meta-range as additional support for EPA's population attributable risk range. The meta-analysis provides continued support for an association between exposure to chlorinated surface water and bladder cancer.

EPA requests comment on the use of a meta-estimated odds ratios to estimate national population attributable risk for the purpose of supporting the benefit analysis for this rule, either based specifically on the Villanueva *et al.* publication or on the application of a similar approach. EPA also solicits comments and suggestions for use of the combined dose-response regression slope coefficient associated with the increased risk of bladder cancer for each additional year's exposure to DBPs in drinking water for estimating the drop in risk associated with a reduction in DBPs as part of the benefit analysis of this rule. EPA provides further discussion and solicitation of comment on how the slope factor might further be considered in estimating the benefits of this rule in the economic section of this preamble.

b. *New colon cancer studies.* Colorectal cancer is the third most common type of new cancer cases and deaths in both men and women in the U.S. It is estimated that 148,300 new colorectal cancer cases will be diagnosed in 2002, with 56,600 resulting in deaths (American Cancer Society, 2002). Human epidemiology studies on chlorinated surface water have reported associations with colorectal cancer. Since the Stage 1 DBPR, two new human epidemiology studies (Yang *et al.* 1998 and King *et al.* 2000b) have been conducted to investigate the relationship between colon cancer and exposure to chlorinated surface water. Yang *et al.* 1998 did not identify an association between consumption of chlorinated drinking water and colon cancer. The King *et al.* (2000b) study found evidence of a DBP association with colon cancer among males, but no association was observed among females.

Similarity of effects reported in animal toxicity and human epidemiology studies strengthen the weight of evidence for an association between DBP exposure and colon cancer. Effects observed in animal studies which included tumors in BDCM exposed rats and mice at several sites (NTP 1987); colon tumors in bromoform exposed rats (NTP 1989); and development of aberrant crypt foci, a preneoplastic lesion of colon cancer in animals exposed to DBP mixtures (DeAngelo *et al.* 2002), are comparable to observations in some cancer epidemiological studies showing an association with colorectal cancer and consumption of chlorinated water (King *et al.* 2000b).

Even with the additional study showing an association, the epidemiological database on colon cancer as a whole is not as strong as that for bladder cancer. However, this new study increases the weight of evidence of an association between DBP exposure

and colon cancer. The Stage 1 DBPR (USEPA 1998c) includes additional discussion of colon cancer risks associated with DBP exposure.

c. *New rectal cancer studies.* The evidence for an association between DBPs and rectal cancer is stronger than for colon cancer. Yang *et al.* (1998) and Hildesheim *et al.* (1998) both found associations between chlorinated drinking water exposure and rectal cancer, and the associations had a similar magnitude in both sexes. Hildesheim *et al.* also found an association in both sexes with lifetime average THM concentration. The consistency of the dose-response trends, the consistency between sexes, and the apparent control of important potential confounders in this study all support the observed associations.

d. *Other cancers.* Two new human epidemiology studies support the possibility of an association between DBPs and kidney cancer. Yang *et al.* (1998) found a positive association for both males and females between consumption of chlorinated drinking water and kidney cancer. Koivusalo *et al.* (1998) found a small, statistically significant, exposure-related excess risk for kidney cancer for males. The association for females was not significant in the Koivusalo *et al.* 1998 study. The current database for this endpoint of cancer, however, is insufficient to conclude an association.

Cantor *et al.* (1999) studied brain cancer, focusing on gliomas. None of the exposure variables were related to brain cancer among females, but males showed a statistically significant, monotonically increasing risk associated with duration of exposure to chlorinated surface water. This study suggests a possible association between chlorination byproducts and gliomas; however, the evidence from this study is not strong enough to support a conclusion of a causal association.

Infante-Rivard *et al.* (2001) conducted a population-based case-control study in Quebec Province, Canada, to examine possible associations between childhood acute lymphoblastic leukemia and THMs. There were no associations with leukemia for any of the exposure indices for total THM, or specific THMs. Therefore, the study does not provide evidence of an association between any of the exposure variables and childhood leukemia.

3. Review of the Cancer Epidemiology Literature (WHO 2000)

The International Programme on Chemical Safety (IPCS) report on disinfectants and disinfection byproducts (WHO 2000) concludes that results of analytical epidemiological cancer studies are insufficient to support a causal relationship for bladder, colon, rectal, or any other cancer and chlorinated drinking water or THMs. The report notes that there is better evidence for an association between exposure to chlorinated surface water and bladder cancer than for other types of cancer. The WHO also concludes that based on the large number of people exposed to chlorinated drinking water, there is a need to address this potential health concern.

E. Cancer and Other Toxicology

Few new cancer toxicology studies have been completed since the Stage 1 DBPR was finalized in December 1998. The information provided in the following sections adds to the toxicology database and provides additional support for the Stage 2 DBPR to control DBP peaks (e.g, high TTHM and HAA5 levels) throughout distribution systems, but does not change the quantitative assessment of the MCLGs.

1. EPA Criteria Documents

To date, EPA has established lifetime cancer risk levels for four DBPs (bromoform, bromodichloromethane, bromate, and dichloroacetic acid) classified as ''probable'' carcinogens, as promulgated in the Stage 1 DBPR and reported in the Integrated Risk Information System (IRIS). Although researchers have continued to assess the cancer risks of DBPs, there has been little change in the overall DBP carcinogenicity database since the Stage 1 DBPR.

The most significant new publication since the Stage 1 DBPR was a study of DCAA tumorigenicity in mice by DeAngelo *et al.* (1999). The Agency has used the data from this study to revise the slope factor for DCAA and a drinking water 10^{-6} lifetime cancer risk concentration. The slope factor is a measure of the potency of a carcinogen while the 10^{-6} lifetime cancer risk concentration provides an estimate of the concentration of a contaminant in drinking water that is associated with an estimated excess lifetime cancer risk of one in a million (Table III–3).

Another significant advancement beyond the Stage 1 DBPR was the evaluation of the chloroform tumorigenicity data on the basis of its nonlinear mode of action following the draft 1999 proposed Guidelines for Carcinogen Risk Assessment (USEPA 1999a). The new chloroform assessment became available on IRIS (2001) in October, 2001 (see section V for a more detailed discussion).

The Criteria Documents for bromoform, bromodichloromethane, dibromochloromethane, and dichloroacetic acid that support the Stage 2 proposal include cancer slope factors and 10^{-6} lifetime cancer risk concentrations that have been modified from their Stage 1 values in order to reflect the methodology proposed in the 1996/1999 draft cancer guidelines (USEPA 1999a) (Table III–3). These include the values based on the Maximum Likelihood Estimate of the dose producing effects in 10 percent of the animals (ED_{10}) and from the lower 95 percent confidence bound on that value (LED_{10}). Except for dibromochloromethane, which is classified as a possible human carcinogen, the DBPs in Table III–3 (and bromate as noted previously) are classified as probable human carcinogens.

TABLE III—3.—QUANTIFICATION OF CANCER RISK

Disinfection byproduct	Risk factors from LED_{10}		Risk factors from ED_{10}	
	Slope factor $(mg/kg/day)^{-1}$	10^{-6} Risk concentration (mg/L)	Slope factor $(mg/kg/day)^{-1}$	10^{-6} Risk concentration (mg/L)
Bromodichloromethane	0.034	0.001	0.022	0.002
Bromoform	0.0045	0.008	0.0034	0.01
Dibromochloromethane	0.04	0.0009	0.017	0.002
Dichloroacetic Acid	0.048	0.0007	0.014	0.003

EPA believes that it is important to pursue additional research on cancer from DBPs. EPA has several ongoing studies in addition to a collaboration with the National Toxicology Program of the National Institute of Environmental Health Sciences. More information on EPA's toxicology research program can be found at *http://www.epa.gov/nheerl.*

2. Other Byproducts with Carcinogenic Potential

a. *3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone) (MX)—multisite cancer.* MX is a byproduct of chlorination that is typically found at very low concentrations (approximately <0.000067 mg/L) in drinking water. The information available on MX was recently compiled in the *Quantitative Cancer Assessment for MX and chlorohydroxyfuranones* (USEPA 2000i). Overall, the weight of evidence indicates that MX is a direct-acting genotoxicant in mammals, with the ability to induce tumors in multiple sites. The primary sites for tumor formation are the thyroid and liver.

b. *N-nitrosodimethylamine (NDMA)—multisite cancer.* Health effects data indicate that NDMA is a probable human carcinogen, as described on IRIS (1991). Risk assessments have estimated that the 10^{-6} lifetime cancer risk level is 0.000007 mg/L based on induction of tumors at multiple sites. Recent studies have produced new information on the occurrence and mechanism of formation of NDMA but there is not enough information at this time to draw conclusions. More research is underway to determine the mechanism by which NDMA is formed in drinking water, and the extent of its occurrence in chloraminated systems.

3. Other Toxicological Effects

The Agency has modified the reference dose (RfD) values for 2 of the chlorinated acetic acids since the Stage 1 DBPR. Under the Stage 1 DBPR there was no established RfD for monochloroacetic acid (MCAA). Data from a drinking water exposure study of MCAA in rats by DeAngelo *et al.* (1997) were used to establish an RfD of 0.004 mg/kg/day based on observed increases in spleen weight. Data from DeAngelo (1997) were also used to calculate a new RfD of 0.03 mg/kg/day for trichloroacetic acid (TCAA) based on observed effects on body weight and liver effects. Detailed discussions of the new reference doses are located in section V of this preamble.

4. WHO Review of the Cancer Toxicology Literature (2000)

The IPCS report on Disinfectants and Disinfection Byproducts (WHO 2000) emphasizes that the bulk of the toxicology data focuses primarily on carcinogenesis. The Task Group found BDCM to be of particular interest because it produces tumors in both rats and mice at several sites. Although the HAAs appear to be without significant genotoxic activity, the brominated HAAs appear to induce oxidative damage to DNA, leading to tumor formation.

F. Conclusions Drawn From the Cancer Epidemiology and Toxicology

EPA believes that the cancer epidemiology and toxicology databases provide important information that contributes to the weight of evidence evaluation of the potential health risks from exposure to chlorinated drinking water. At this time the cancer epidemiology studies are insufficient to establish a causal relationship between exposure to chlorinated drinking water and cancer, but EPA does believe there is a potential association. The current database is sufficient for quantitative analysis on the endpoint of bladder cancer, as presented previously in the PAR analysis.

The association between DBP exposure and colon cancer remains more tenuous than the link to bladder cancer, although similarity of effects reported in animal toxicity and human epidemiology studies strengthens the weight of evidence for an association between DBP exposure and colon cancer. Studies finding potential relationships between exposure to chlorinated drinking water and rectal, kidney, and brain cancer also add to the weight of evidence for a public health concern. EPA believes that the overall cancer epidemiology and toxicology data support the decision to pursue additional DBP control measures as reflected in the Stage 2 DBPR.

G. Request for Comment

EPA requests comment on the conclusions drawn from the new health information summarized in this section. EPA requests comment on the weight of evidence evaluation of the potential reproductive and developmental hazards from DBPs and its potential implications for the regulatory provisions for the final Stage 2 DBPR. EPA solicits any additional data on the reproductive or developmental effects from DBPs that need to be considered for the final Stage 2 DBPR.

EPA requests comment on EPA's conclusions regarding cancer epidemiology and toxicology, and the new studies discussed in today's proposal. EPA solicits any additional cancer epidemiology and toxicology data that need to be considered for the final Stage 2 DBPR.

EPA also solicits any health information available to further assess risk to sensitive subpopulations, especially children and the elderly.

IV. DBP Occurrence Within Distribution Systems

New information on the occurrence of DBPs in distribution systems raises issues about the protection provided by the Stage 1 DBPR. This section presents the new information used to identify key issues and to support the development of the Stage 2 DBPR. For a more detailed discussion see the *Stage 2 Occurrence Assessment for Disinfectants and Disinfection Byproducts* (USEPA 2003o).

Under the Stage 1 DBPR, compliance with the DBP MCLs is determined by averaging, annually and system-wide, all DBP measurements. The following discussion shows that compliance based on system averages of DBP concentrations allows a significant number of sampling locations within distribution systems to have DBP levels above the MCLs. These peak DBP occurrences are masked by averaging with lower distribution system occurrence levels. The populations served by portions of the distribution system with higher DBP concentrations are not receiving the same level of health protection.

The new information also shows that the highest DBP levels often do not occur at distribution system sites identified as representing maximum residence time. The information further shows that the highest TTHM and HAA5 levels often do not occur at the same site within the distribution system. These two findings suggest that it is appropriate to reevaluate the Stage 1 DBPR compliance monitoring sites in order to target those sites with high DBP levels. EPA believes that distribution system compliance monitoring sites need to be reevaluated to ensure identification of sites that reflect both high TTHM and HAA5 occurrence.

A. Data Sources

1. Information Collection Rule Data

The Information Collection Rule (USEPA 1996a) established monitoring and data reporting requirements for large public water systems. Under the Information Collection Rule, systems serving at least 100,000 people were required to conduct DBP and DBP-

related monitoring. The 18 months of required monitoring, which began in July 1997 and ended in December 1998, applied to 296 public water systems (500 treatment plants).

The Information Collection Rule data show the national occurrence of: (1) Influent water quality parameters; (2) primary and secondary disinfectant use by the large plants; (3) occurrence of DBPs and DBP precursors in treatment plants, finished waters, and distributions systems; (4) microbial occurrence (in subpart H systems only); and (5) treatment plant monthly operation, and initial as well as final treatment plant design. The data were gathered after the Stage 1 DBPR was finalized (USEPA 1998c) but well before systems were required to meet Stage 1 DBPR requirements.

The Information Collection Rule required a significant investment for the water treatment industry, as well as for the EPA to analyze the data. Overall, the occurrence and treatment data collected under the Information Collection Rule, excluding microbial data, was estimated to cost systems $54 million (USEPA 1996a). In addition, systems using source waters with high DBP precursor levels were required to conduct bench and pilot studies to evaluate the effectiveness of granular activated carbon (GAC) and membrane technology to control for DBPs. The estimated cost for these studies totaled approximately $57 million (USEPA 1996a).

In addition to the analysis of DBPs in distribution systems, EPA used occurrence data from the Information Collection Rule to confirm selection of TTHM and HAA5 as appropriate contaminants for monitoring DBPs. EPA also used occurrence data from the Information Collection Rule to confirm differences in monitoring requirements for systems using surface water versus those using ground water, as stipulated under the Stage 1 DBR. Analysis of the Information Collection Rule data indicates that TTHM and HAA5 comprise on average, across all systems, about 50% of the total mixture of chlorinated DBPs and that TTHM and HAA5 concentrations are much lower and less variable in ground water systems than in surface water systems. These results support the basis for continuing the use of TTHM and HAA5 as indicators for controlling chlorinated DBPs. The data also reconfirmed that ground water systems require less monitoring than surface water systems based on lower and less variable DBP occurrence. For detailed analysis, see *Stage 2 Occurrence Assessment for Disinfectants and Disinfection Byproducts* (USEPA 2003o).

2. Other Data Sources Used To Support the Proposal

Table IV–1 summarizes the data sources other than the Information Collection Rule used to support the Stage 2 DBPR. The data from the Information Collection Rule is from large systems. To validate the conclusions drawn from analysis of the Information Collection Rule for small and medium systems, EPA compared these other data sources with the Information Collection Rule data. EPA found that there are significant similarities between large systems and medium and small systems with regard to source water quality (affecting DBP formation) and use of treatment technologies. Because of these similarities, EPA expects that small and medium systems would find DBP distribution system levels similar to those found in large systems following compliance with the Stage 1 DBPR requirements. For detailed discussion of this analysis, see *Stage 2 Occurrence Assessment for Disinfectants and Disinfection Byproducts* (USEPA 2003o) and *Economic Analysis for the Stage 2 Disinfection Byproducts Rule* (USEPA 2003i).

TABLE IV–1.—SUMMARY OF NON-INFORMATION COLLECTION RULE OCCURRENCE SURVEY DATA

Data source	Data collected	Geographic representation	Number of plants (By population served)
Information Collection Rule Supplemental Survey.	Raw source water-(Large Systems) TOC Raw source water-(Small & Medium Survey Systems) TOC, UV 254, bromide, turbidity, pH, & temperature.	Random national distribution by SW source type [1].	47 serving 100,000 or more. 40 serving 10,000–99,999. 40 serving fewer than 10,000.
WaterStats	Population served and flows Raw source water—Water Quality Parameters (WQPs), Source water type. Finished water-WQPs, TTHM, HAAs Treatment-unit processes, disinfectant used.	Random national distribution.	219 serving 100,000 or more. 623 serving 10,000–99,999. 30 serving fewer than 10,000.
National Rural Water Association Survey (NRWAS).	Population served and flows Raw source water-temperatures, turbidity, pH, and source water type, bromide, TOC, UV 254, alkalinity, calcium, and total hardness. Finished water-residence time estimate, total and individual THMs, individual HAAs and HAA5, HAA6, HAA9,TOC, UV 254, Bromide, Temperature, pH, free and total chlorine residual levels. Treatment-unit processes, disinfectant used.	Random national distribution.	117 serving fewer than 10,000.
State Data-Surface Water ..	Distribution system TTHM occurrence data.	AK, CA, IL, MN, MS, NC, TX, WA [2].	562 serving fewer than 10,000.
State Data-Ground Water ..	Distribution system TTHM occurrence data.	AK, CA, FL, IL, NC, TX, WA [2].	2336 serving fewer than 10,000.
Ground Water Supply Survey.	TOC and TTHM (one sample for each parameter at the entry point to distribution system.)	Random national distribution.	979 total.

[1] Source type designations include flowing stream and lake/reservoir (Except for 7 large plants pre-selected).

[2] Over 50 percent of each State's systems are represented. EPA believes that the data reasonably represent a full range of source water quality in small systems at the national level.

B. DBPs in Distribution Systems

EPA wanted to understand DBP occurrence in distribution systems likely to exist after implementation of the Stage 1 DBPR. Such an understanding would enable EPA to recognize options on how to improve protection under the Stage 2 DBPR. The analysis of occurrence data to support the Stage 2 DBPR is complicated because available national occurrence data do not reflect the changes in occurrence resulting from the implementation of the Stage 1 DBPR. Many utilities have only recently changed their treatment to comply with the Stage 1 DBPR (subpart H systems serving 10,000 people or more were required to comply beginning January 2002) or are about to make changes in treatment to comply with this rule (subpart H systems serving fewer than 10,000 people and ground water systems are required to comply beginning January 2004).

To address the above issue, EPA evaluated Stage 1 DBPR implications by using Information Collection Rule data from plants that would not exceed the Stage 1 DBPR TTHM and HAA5 MCLs as an annual average. The TTHM and HAA5 data consist of quarterly measurements in four locations in distribution systems associated with each Information Collection Rule treatment plant. Two samples were collected at sites representing average residence time (AVG1 and AVG2), one sample at a site intended to represent the maximum residence time (MAX), and one sample was reported as a distribution system equivalent (DSE). The DSE sample was generally representative of average residence times. EPA believes that the monitoring locations of the Information Collection Rule, while not necessarily being the same as the Stage 1 DBPR compliance monitoring sites, provide a close approximation of monitoring under the Stage 1 DBPR. EPA recognizes, however, that data for plants that are in compliance with Stage 1 MCLs even without installing additional treatment (perhaps because of low source water TOC) are not necessarily reflective of plants that make treatment changes to comply with the Stage 1 DBPR.

1. DBPs Above the MCL Occur at Some Locations in a Substantial Number of Plants

Figure IV–1 compares the TTHM running annual average (RAA) levels with the single highest TTHM concentration in the distribution system. Twenty one percent (60 of 290) of the Information Collection Rule plants had single TTHM concentrations higher than the 0.080 mg/L MCL. Figure IV–2 makes the same comparison for HAA5. Fourteen percent (40 of 290) of the plants meeting the Stage 1 DBPR MCL had single HAA5 concentrations higher than the 0.060 mg/L MCL. In systems with a low RAA for TTHM and HAA5, the highest single TTHM and HAA5 values are generally not much higher than the respective Stage 1 DBPR MCLs. However, as the RAAs increase, there is a greater likelihood of having peak levels above the MCLs. As the RAAs approach the Stage 1 DBPR MCLs, some of the distribution system single highest concentrations approach levels that are double the Stage 1 DBPR MCLs.

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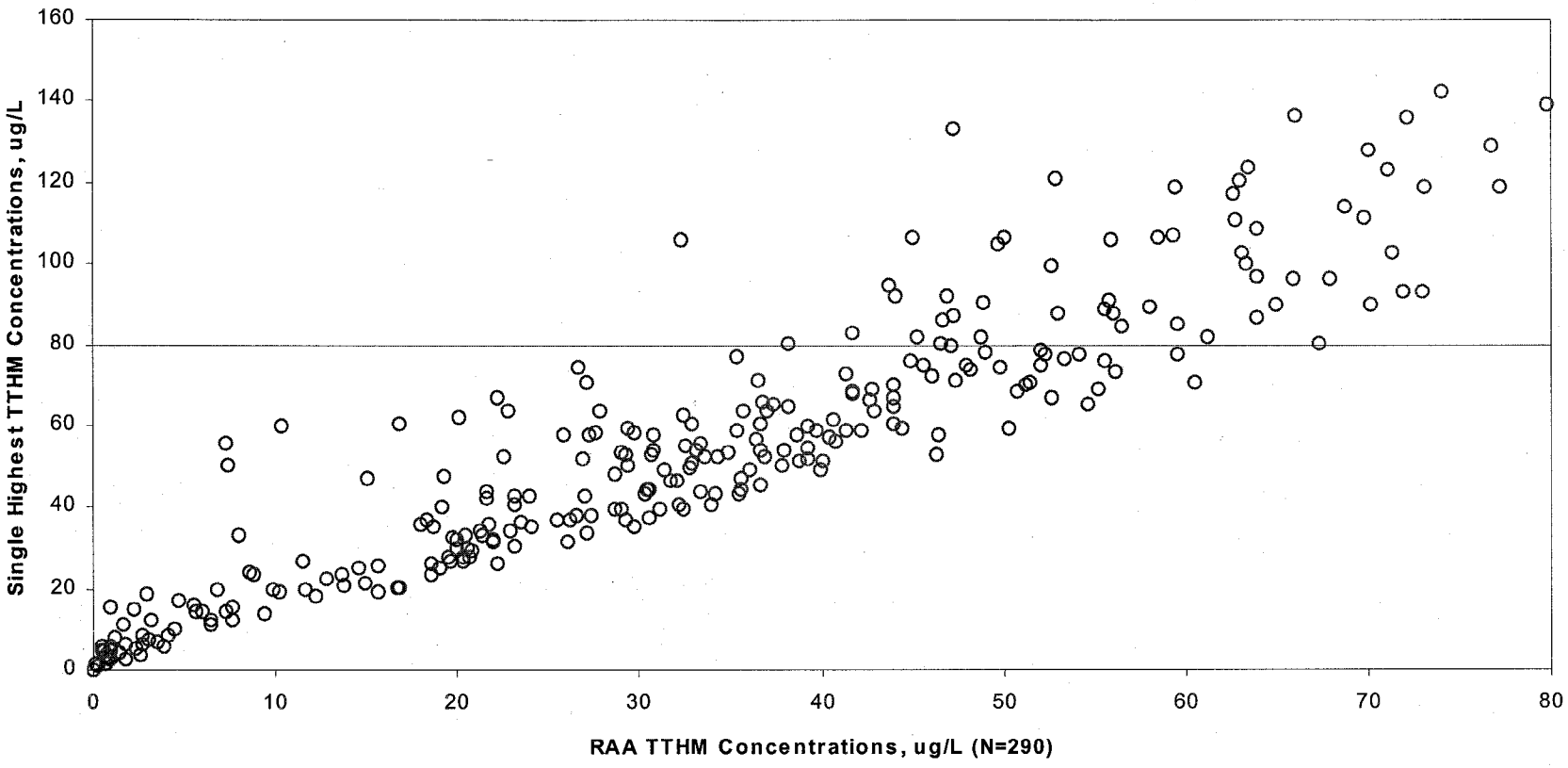
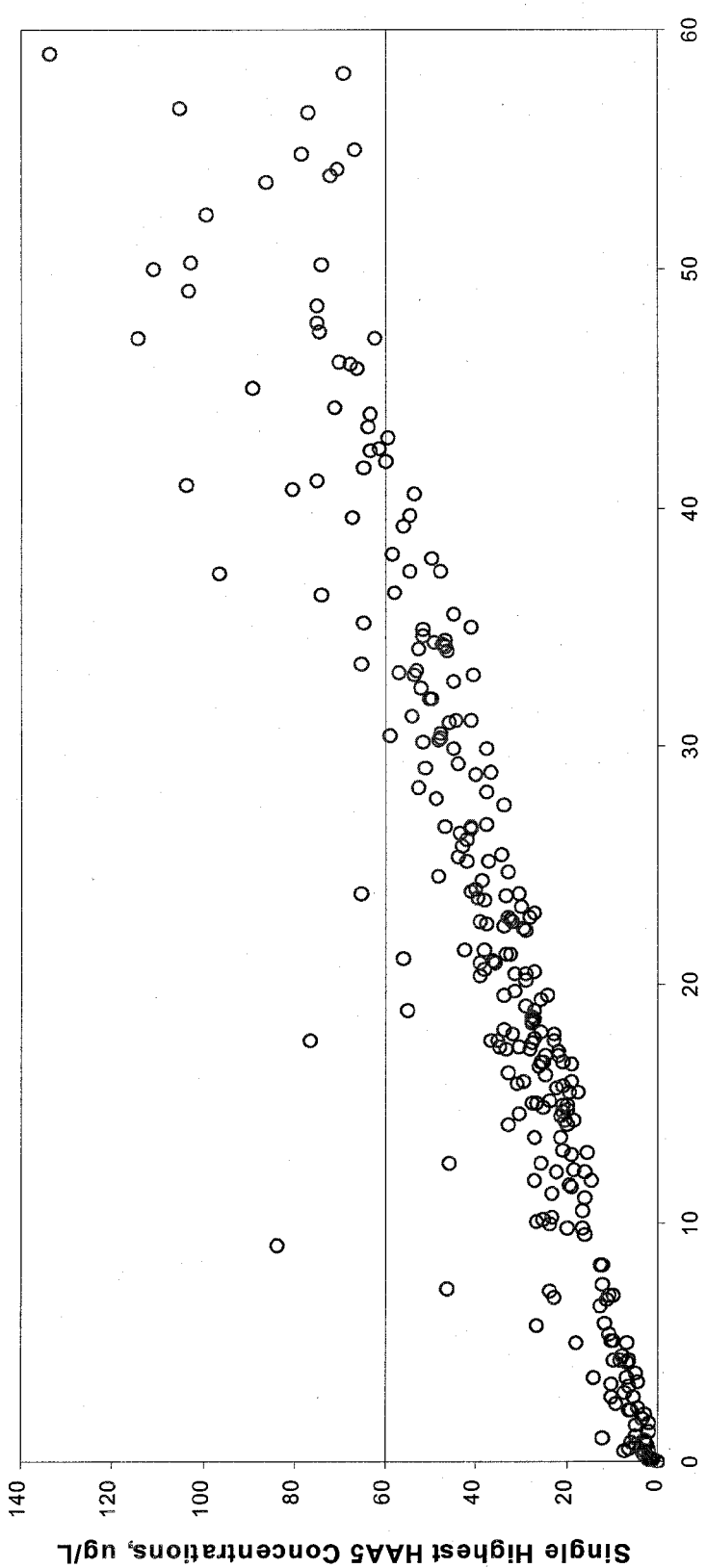
Figure IV-1. Single Highest TTHM Concentrations Versus TTHM RAA Concentrations for Plants Meeting Stage 1 DBPR MCLs



[1] Includes only the Information Collection Rule plants with at least 3 quarters of data and with each quarter having at least 3 sampling locations for both TTHM and HAA5 during the last 4 quarters of the Information Collection Rule sampling period.

Figure IV-2 Single Highest HAA5 Concentrations Versus HAA5 RAA Concentrations for Plants Meeting Stage 1 DBPR MCLs



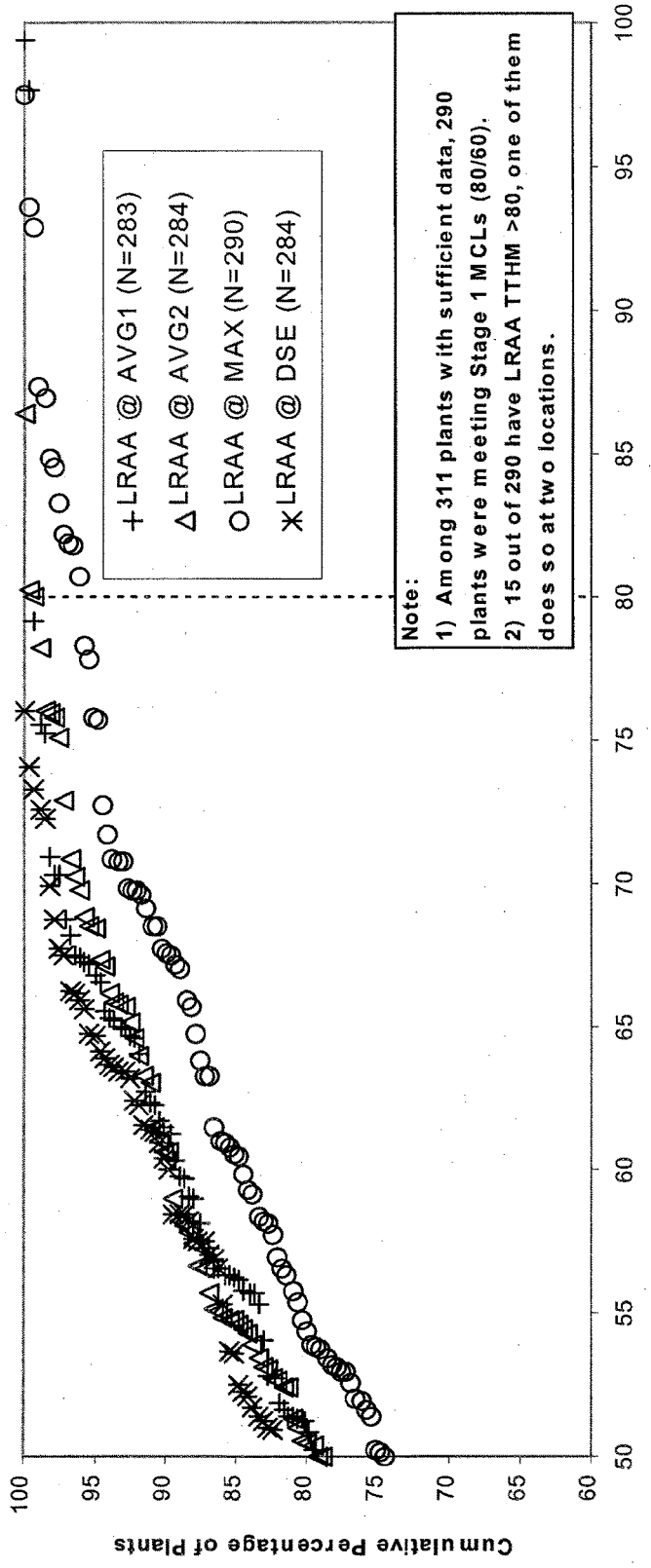
[1] Includes only the Information Collection Rule plants with at least 3 quarters of data and with each quarter having at least 3 sampling locations for both TTHM and HAA5 during the last 4 quarters of the Information Collection Rule sampling period.

2. Specific Locations in Distribution Systems Are Not Protected to MCL Levels

Data from the Information Collection Rule show that the RAA compliance calculation may allow specific locations in a distribution system to regularly receive water with DBP levels that exceed the MCL. Figure IV–3 shows that five percent of plants (15 out of 290) had one or more locations that, on average, exceeded 0.080 mg/L as a TTHM LRAA for that same year. One of the 15 plants that exceeded a TTHM LRAA of 0.080 mg/L did so at two locations. Of the 15 plants, the highest LRAA was between 0.080 and 0.090 mg/L at 10 plants, and between 0.090 and 0.100 mg/L at 5 plants. Customers served at these locations regularly received water with TTHM concentrations somewhat higher than the MCL.

Figure IV–4 shows similar results based on Information Collection Rule HAA5 data. Three percent of plants (eight of 290) exceeded 0.060 mg/L as an LRAA, and three of these eight plants did so at two or three locations. Of the 8 plants, the highest LRAA was between 0.060 and 0.070 mg/L at 5 plants, and between 0.070 and 0.075 mg/L at 3 plants. Among the 290 plants in the Information Collection Rule database meeting the Stage 1 MCLs, 19 plants have a maximum TTHM LRAA of 0.080 mg/l or greater or a maximum HAA5 LRAA of 0.060 mg/l or greater (four plants exceeded both MCLs), though in no case did DBP levels at a given location consistently exceed the MCL by more than 20%.

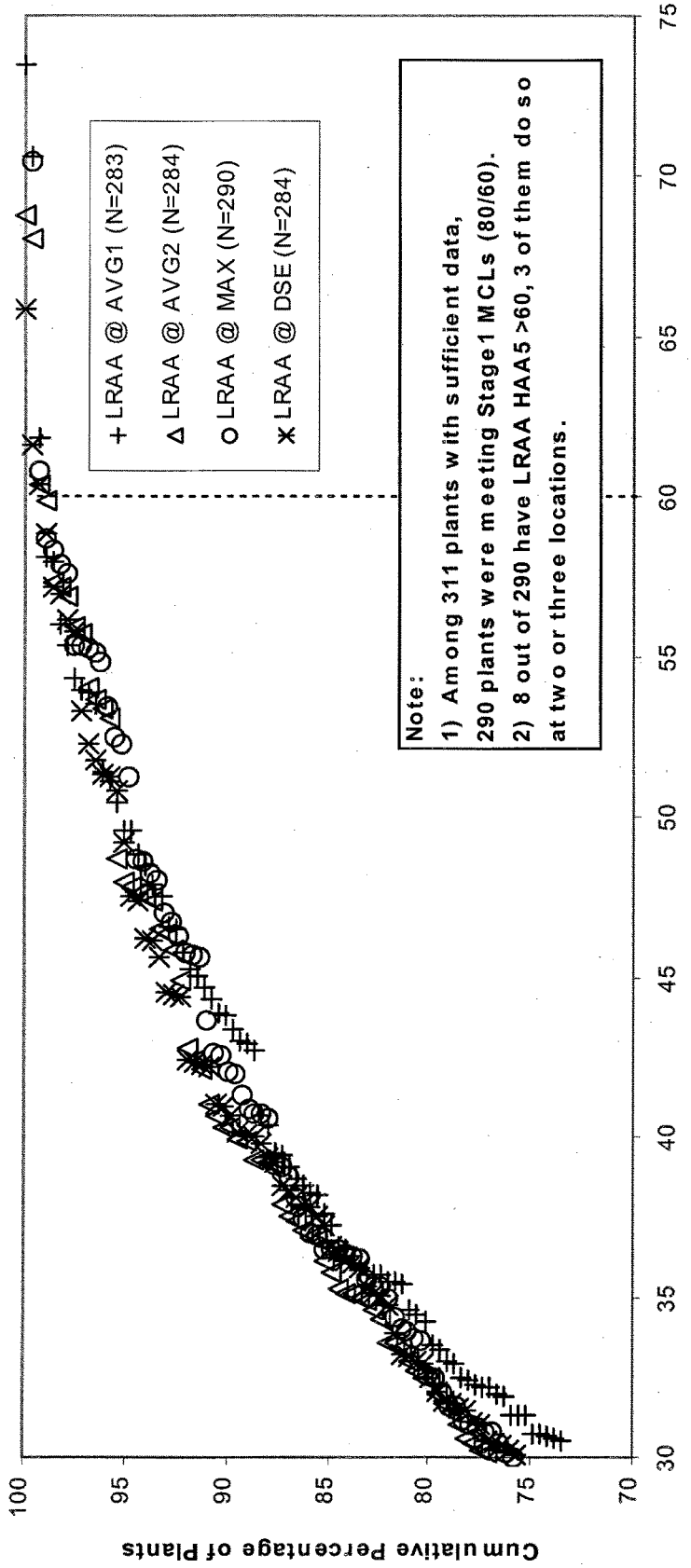
Figure IV-3. Cumulative Percentage of TTHM LRAAs for Surface Water Plants[1,2]



The data shown are for surface water plants that would not exceed the Stage 1 DBPR TTHM and HAA5 MCLs when calculated using the last 4 quarters of Information Collection Rule data.

[2] Includes only the Information Collection Rule plants with at least 3 quarters of data and with each quarter having at least 3 sampling locations for both TTHM and HAA5 during the last 4 quarters of the Information Collection Rule sampling period.

Figure IV-4. Cumulative Percentage of HAA5 LRAAs for Surface Water Plants[1,2].



[1] The data shown are for surface water plants that would not exceed the Stage 1 DBPR TTHM and HAA5 MCLs when calculated using the last 4 quarters of Information Collection Rule data.

[2]Includes only the Information Collection Rule plants with at least 3 quarters of data and with each quarter having at least 3 sampling locations for both TTHM and HAA5 during the last 4 quarters of the Information Collection Rule sampling period.

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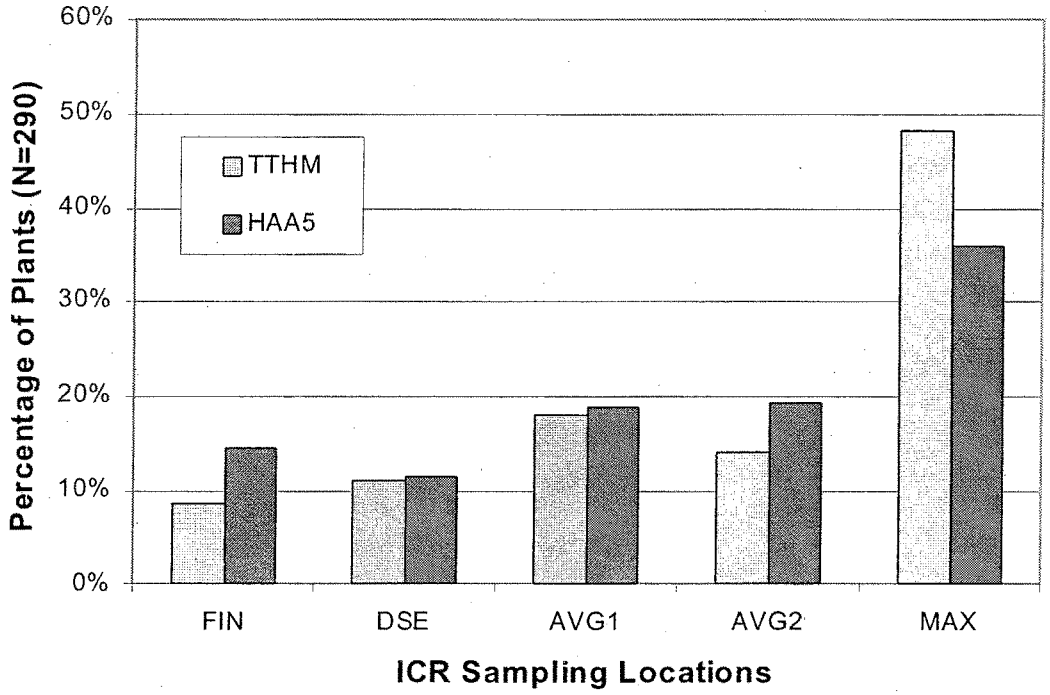
3. Stage 1 DBPR Maximum Residence Time Location May Not Reflect the Highest DBP Occurrence Levels

The 1979 TTHM rule and Stage 1 DBPR monitoring locations must include a site reflection maximum residence time in the distribution system with the intent of capturing the highest DBP levels in the distribution system. The Information Collection rule referred to this specific location as MAX. The Information Collection rule data indicate two important results: (1) that monitoring locations identified as the maximum residence time locations often did not represent those locations with the highest DBP levels and (2) the highest TTHM and HAA5 level often occurred at different points in the distribution system.

Figure IV–5 illustrates that the highest TTHM and HAA5 LRAAs could be at any of the four Information Collection Rule sample locations in the distribution system or, in some cases, at the finished water location. Fifty percent of the plants evaluated have the highest TTHM LRAA concentration occurring at a site other than the maximum residence time monitoring site. over 60% of plants evaluated had the highest HAA5 LRAA at a location other than the maximum residence time monitoring site.

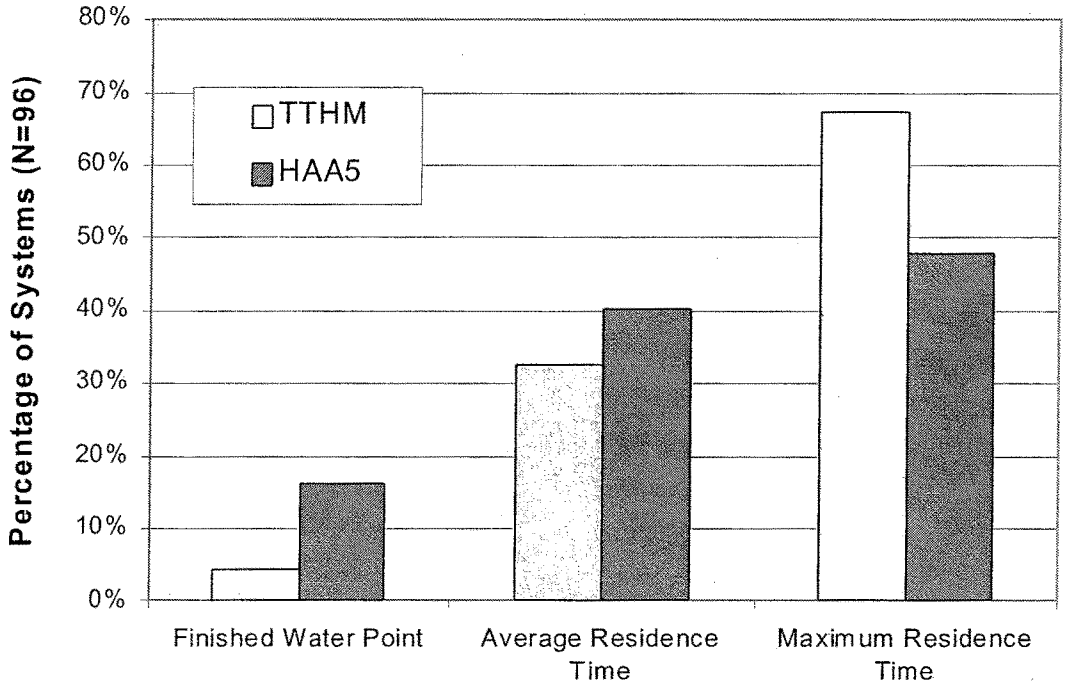
Figure IV–6, based on data from the National Rural Water Survey (NRWS), indicates that systems serving fewer than 10,000 people also frequently have their highest TTHM and HAAS levels at locations other than those intended to represent maximum residence time. The occurrence patterns indicated in Figures IV–5 and IV–6 may be due to several factors, such as HHA5 degrading over time in the distribution system, maximum residence time monitoring sites not actually representing the maximum residence time, or that using a simple estimation of maximum residence time cannot characterize a complex distribution system.

Figure IV-5. Frequency at Which Highest TTHM or HAA5 Locational Annual Average Concentrations Occurred at Each Information Collection Rule Sampling Location for plants meeting Stage 1 MCLs[1]



[1] Includes only the Information Collection Rule plants with at least 3 quarters of data and with each quarter having at least 3 sampling locations for both TTHM and HAA5 during the last 4 quarters of the Information Collection Rule sampling period.

Figure IV-6 Frequency at Which Highest TTHM or HAA5 Locational Annual Average Concentrations Occurred at Each Monitoring Location in NRWA Survey



Note: LRAA calculation is based on the results from two sampling events at each of sampling locations in the NRWA survey (winter and summer months). Only plants having monitoring results for both winter and summer months are included in the data set.

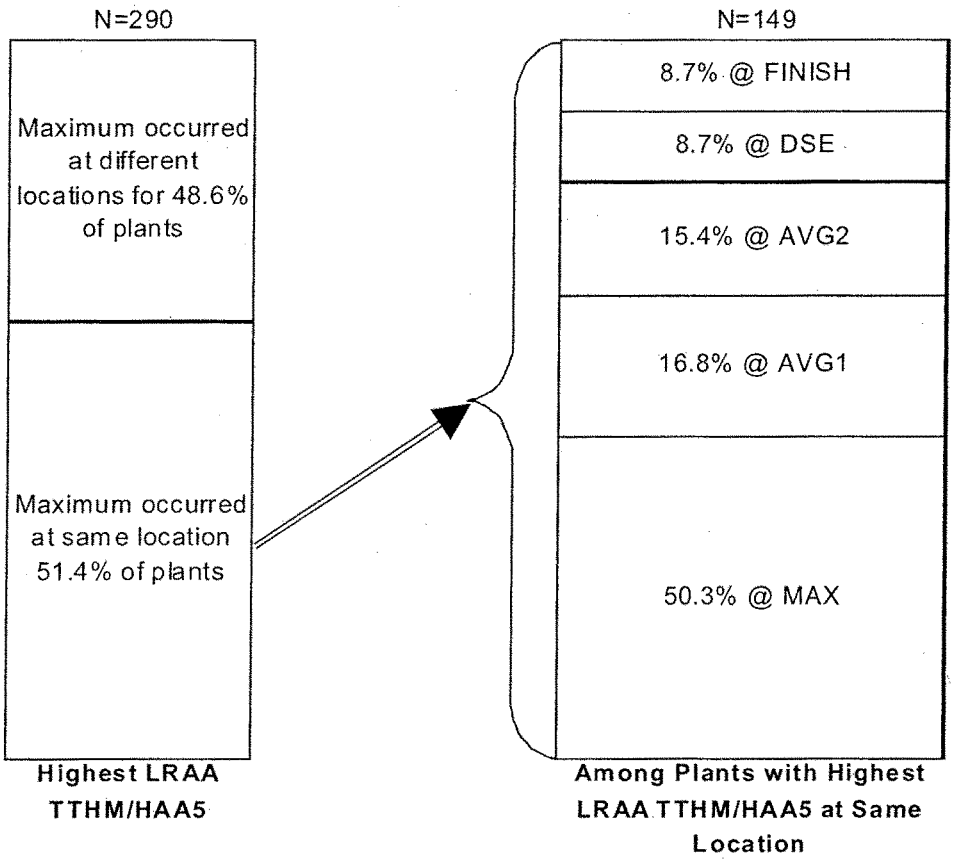
EPA also analyzed whether the highest LRAA for TTHM and HAA5 occurred at the same location. If TTHM and HAA5 occur at the same location rather than different locations, fewer monitoring sites would be needed to represent TTHM and HAA5 occurrence. However, this is not the case. The Information Collection Rule and NRWA data sets, respectively, indicate that 49% and 44% of plants experienced their highest LRAA TTHM and HAA5 concentrations at different locations in the distribution system.

For plants that did have their highest LRAA TTHM and HAA5 concentrations at the same location, it was not necessarily the maximum residence time monitoring location. Figure IV–7 illustrates that for the Information Collection Rule plants with the highest TTHM and HAA5 levels occurring at the same location, the highest TTHM and HAA5 LRAA simultaneously occurred at the maximum residence time monitoring location in 50% of the cases. Figure IV–8 illustrates that for the NRWA plants with the highest TTHM and HAA5 levels occurring at the same location, the highest TTHM and HAA5 LRAA simultaneously occurred at the maximum residence time (MAX) monitoring location in 64% of the cases.

C. Request for Comment

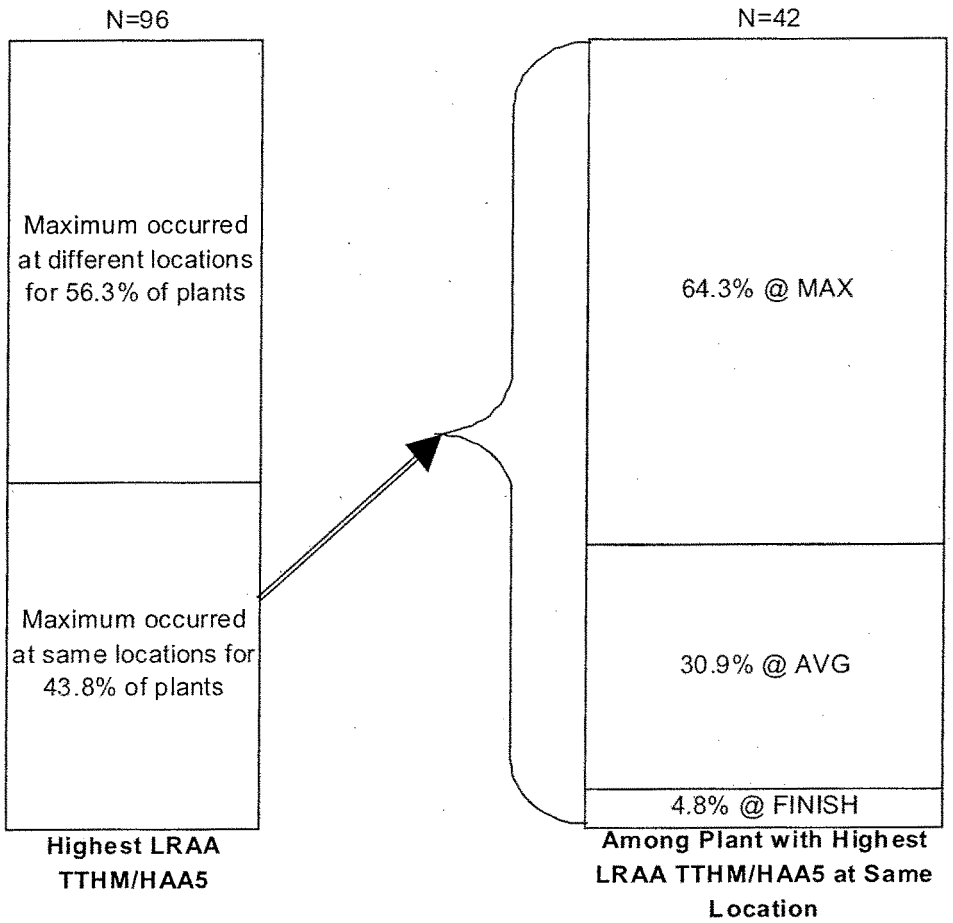
EPA requests comment on the analysis presented in this section. Is EPA's approach for representing post Stage 1 DBPR occurrence appropriate? What other approaches might be used? Are the conclusions that EPA derives from the analysis appropriate?

Figure IV-7. Frequency at Which Highest TTHM/HAA5 LRAAs Occurred at Same Sampling Location (Based on the Information Collection Rule data) for plants meeting Stage 1 MCLs. [1]



[1]Includes only the Information Collection Rule plants with at least 3 quarters of data and with each quarter having at least 3 sampling locations for both TTHM and HAA5 during the last 4 quarters of the Information Collection Rule sampling period.

Figure IV-8. Frequency at Which Highest TTHM/HAA5 LRAAs Occurred at Same Sampling Location (Based on the data from NRWA Survey)



Note: LRAA calculation is based on the results from two sampling events at each of sampling locations in the NRWA survey (winter and summer months). Only plants having monitoring results for both winter and summer months are included in the data set.

V. Discussion of Proposed Stage 2 DBPR Requirements

A. MCLG for Chloroform

1. What Is EPA Proposing Today?

EPA is proposing an MCLG for chloroform of 0.07 mg/L based on a cancer reference dose (RfD), an assumption that a person drinks 2 liters of water per day (the 90th percentile of intake rate for the U.S. population), and a relative source contribution (RSC) of 20 percent. The MCLG is proposed at a level at which no adverse effects on the health of persons is anticipated with an adequate margin of safety. This conclusion is based on toxicological evidence that the carcinogenic effects of chloroform are an ultimate consequence of sustained tissue toxicity. The MCLG is set at a daily dose for a lifetime at which no adverse effects will occur because the sustained tissue toxicity, which is a key event in the cancer mode of action of chloroform, will not occur (USEPA 2001b).

EPA believes that the RfD used for chloroform is protective of sensitive groups, including children. This RfD was developed by the EPA current method for developing RfDs based on animal data. The method is designed to be protective by taking human variability into account and assuming that the average human will be as sensitive as the most responsive animal species. EPA's understanding of the mode of action for chloroform does not indicate a uniquely sensitive subgroup or an increased sensitivity in children.

2. How Was This Proposal Developed?

a. *Background.* EPA proposed a zero MCLG for chloroform in the 1994 Stage 1 DBPR proposal (USEPA 1994b). Following the proposal, numerous toxicological studies on chloroform were published and were discussed in two Notices of Data Availability (NODAs) (USEPA 1997a; USEPA 1998e). The 1998 NODA presented substantial scientific data related to the mode of action as part of the chloroform risk assessment and requested comment on a chloroform MCLG of 0.3 mg/L that reflected a nonlinear mode of action. After considering comments on the NODAs, EPA determined that further deliberations with the Science Advisory Board (SAB) and stakeholders were needed before changing the MCLG for chloroform. Thus, EPA promulgated a chloroform MCLG of zero in the final Stage 1 DBPR (USEPA 1998c) and committed to conducting additional deliberations with the SAB and factoring the SAB's review into the Agency's Stage 2 DBPR rulemaking

process. The Agency consulted with the SAB in October 1999 (USEPA 2000f).

The Stage 1 DBPR MCLG of zero for chloroform was challenged, and the U.S. Court of Appeals for the District of Columbia Circuit issued an order vacating the zero MCLG (*Chlorine Chemistry Council and Chemical Manufacturers Association* v. *EPA,* 206 f.3d 1286 (D.C. Circuit 2000)). EPA committed to the Court to propose a non-zero MCLG for chloroform in the upcoming proposed Stage 2 Disinfectants and Disinfection Byproducts Rule. EPA removed the MCLG for chloroform from its Stage 1 DBP NPDWR (USEPA 2000e). No other provision of the Stage 1 DBPR was affected.

b. *Basis of the new chloroform MCLG.* Based on an analysis of all the available scientific data on chloroform discussed in more detail below, EPA believes that chloroform dose-response is nonlinear and that chloroform is likely to be carcinogenic only under high exposure conditions. EPA's assessment of the cancer risk associated with chloroform exposure (USEPA 2001b) uses the principles of the 1999 EPA Proposed Guidelines for Carcinogen Risk Assessment (USEPA 1999a).

The Proposed Guidelines for Carcinogen Risk Assessment, as reviewed by the public and the EPA SAB, reflect new science and are consistent with, and an extension of, the existing 1986 Guidelines for Carcinogen Risk Assessment (USEPA 1986). The 1986 guidelines provide for departures from default assumptions such as low dose linear extrapolation. For example, the 1986 EPA guidelines reflect the position of the Office of Science and Technology Policy (OSTP) that (OSTP 1985; Principle 26) ''[N]o single mathematical procedure is recognized as the most appropriate for low-dose extrapolation in carcinogenesis. When relevant biological evidence on mechanisms of action exists (e.g, pharmacokinetics, target organ dose), the models or procedure employed should be consistent with the evidence.'' The 1985 guidelines go on to state ''The Agency will review each assessment as to the evidence on carcinogenesis mechanisms and other biological or statistical evidence that indicates the suitability of a particular extrapolation model.''

i. *Mode of action.* EPA has fully evaluated the science on chloroform and concludes that chloroform is likely to be carcinogenic to humans under high exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissue; chloroform is not likely to be carcinogenic to humans at a dose level that does not cause cytotoxicity and cell regeneration (USEPA 1998e, USEPA 1998b, USEPA 2001b).

Chloroform's carcinogenic potential is indicated by animal tumor evidence (liver tumors in mice and renal tumors in both mice and rats) from inhalation and oral exposure. Data on metabolism, toxicity, mutagenicity and cellular proliferation contribute to an understanding of the mode of carcinogenic action. For chloroform, sustained or repeated cytotoxicity with secondary regenerative hyperplasia precedes, and is a key event for, hepatic and renal neoplasia.

EPA believes that a DNA reactive mutagenic mode of action is not likely to be the predominant influence of chloroform on the carcinogenic process. EPA has concluded that the predominant mode of action involves cytotoxicity produced by the oxidative generation of highly reactive metabolites, followed by regenerative cell proliferation (USEPA 2001b). EPA further believes that the chloroform dose-response is nonlinear. The SAB final report states ''(t)he Subcommittee agrees with EPA that sustained or repeated cytotoxicity with secondary regenerative hyperplasia in the liver and/or kidney of rats and mice precedes, and is probably a causal factor for, hepatic and renal neoplasia'' (USEPA 2000f).

ii. *Metabolism.* The cytochrome P450 isoenzyme CYP 2E1 is the primary enzyme catalyzing chloroform metabolism at low concentrations. Chloroform's carcinogenic effects involve oxidative generation of reactive and toxic metabolites (phosgene and hydrochloric acid [HCl]) and thus are related to its noncancer toxicities (*e.g.*, liver or kidney toxicities). The electrophilic metabolite phosgene could react with macromolecules such as phosphotidyl inositols or tyrosine kinases which in turn could potentially lead to interference with signal transduction pathways (*i.e.*, chemical messages controlling cell division), thus leading to carcinogenesis. Likewise, it is also plausible that phosgene reacts with cellular phospholipids, peptides and proteins resulting in generalized tissue injury. Glutathione, free cysteine, histidine, methionine and tyrosine are all potential reactants for electrophilic agents.

At high concentrations, chloroform may undergo reductive metabolism which forms reactive dichloromethyl free radicals. These free radicals can contribute to lipid peroxidation and cause cytotoxicity.

c. *How the MCLG is derived.* EPA continues to recognize the strength of the science in support of a nonlinear approach for estimating the carcinogenicity of chloroform. This science was affirmed by the Chloroform Risk Assessment Review Subcommittee of the EPA SAB Executive Committee which met on October 27–28, 1999 (USEPA 2000f). The SAB Subcommittee agreed that the nonlinear approach is most appropriate for the risk assessment of chloroform.

Nonzero MCLGs are scientifically and statutorily supported. The statute requires that the MCLG be set where no known or anticipated adverse effects occur, allowing for an adequate margin of safety (56 FR 3533; USEPA 1991b). Historically, EPA established MCLGs of zero for known or probable human carcinogens based on the principle that any exposure to carcinogens might represent some finite level of risk. If there is substantial scientific evidence, however, that indicates there is a ''safe threshold'', then a nonzero MCLG can be established with an adequate margin of safety (56 FR 3533; USEPA 1991a)).

EPA would ideally like to use the delivered dose (i.e., the amount of key chloroform metabolites that actually reach the liver and cause cell toxicity) for calculating an RfD to support the MCLG. However, the required toxicokinetic data are not currently available. Thus, the RfD is calculated using the applied dose (i.e., the amount of chloroform ingested). The RfD is based on both the benchmark dose and the traditional no observed adverse effect level/lowest observed adverse effect level (NOAEL/LOAEL) approaches for hepatotoxicity in the most sensitive species, the dog. The MCLG is based on the RfD and calculated as follows:

$$\text{MCLG} = \frac{\text{RfD} \times \text{body weight} \times \text{RSC}}{\text{daily water consumption}}$$

i. *Reference dose.* The RfD for chloroform was estimated based on noncancer effects using both the benchmark dose and the traditional NOAEL/LOAEL approaches. For benchmark analysis, five relevant data sets including target organ toxicity, labeling index, histopathology in rodents, and liver toxicity in dogs (Heywood 1979) were evaluated. The effects seen in dogs are considered to be early signs of liver toxicity, preceding cytotoxicity, cytolethality and regenerative hyperplasia. Thus, the Heywood (1979) study, provides the most sensitive end point in the most sensitive species and is the most appropriate basis for the RfD.

The 95% confidence lower bound on the dose associated with a 10% extra risk (LED10) is based on the prevalence of animals demonstrating liver toxicity. After an exposure adjustment to the LED10 (1.2 mg/kg/day), an RfD of 0.01 mg/kg/day was calculated using an overall uncertainty factor of 100 (10 for interspecies extrapolation and 10 for protection of sensitive individuals) (USEPA 2001b).

Coincidentally, the benchmark dose and the traditional NOAEL/LOAEL approaches yield the same RfD number (USEPA 2001b). The NOAEL/LOAEL approach is also based on the Heywood study (1979) which had a LOAEL of 15 mg/kg/day for evidence of liver toxicity. After an exposure adjustment to the LOAEL (yielding 12.9 mg/kg/day), an RfD of 0.01 mg/kg/day was calculated using an overall uncertainty factor of 1000 (10 for interspecies extrapolation, 10 for protection of sensitive individuals, and 10 for using a LOAEL instead of a NOAEL) (USEPA 2001b).

ii. *Relative source contribution.* Another factor in determining the MCLG is the relative source contribution (RSC). The RSC is used when the MCLG is set at a level above zero. Its purpose is to ensure that the contribution to exposure from drinking tap water does not cause the lifetime daily exposure of persons to a contaminant to exceed RfD. The RSC is thus a factor used to make sure that the MCLG is protective even if persons are exposed to the contaminant by other routes (inhalation, dermal absorption) or other sources (*e.g.*, food). If sufficient quantitative data are not available on exposure by other routes and sources, EPA has historically assumed that the RSC from drinking water is 20 percent of the total exposure, a value considered protective. If data indicate that contributions from other routes and sources are not significant, EPA has historically assumed a less conservative RSC of 80 percent (54 FR 22,062, 22,069 (May 22, 1989)(USEPA 1989a), 56 FR at 3535 (Jan 30, 1990)(USEPA 1991a), 59 FR 38,668, 38,678 (July 29, 1994)(USEPA 1994b)).

Today, EPA is proposing an assumption of a 20 percent RSC. This is in consideration of data which indicate that exposure to chloroform by other routes and sources of exposure may potentially contribute a substantial percentage of the overall exposure to chloroform.

In the 1998 Stage 1 DBPR NODA, EPA considered an MCLG of 0.3 mg/L that was calculated using an RSC of 80 percent, based on the assumption that most exposure to chloroform is likely to come from ingestion of drinking water. In the final Stage 1 DBPR, EPA reconsidered this assumption in response to comments and in the light of data which indicate that exposure to chloroform by inhalation and dermal exposure may potentially contribute a substantial percentage of the overall exposure to chloroform depending on the activity patterns of individuals (USEPA 1998e) *e.g.,* during showering, bathing, swimming, boiling water, clothes washing, and dishwashing. There is also potential exposure to chloroform by the dietary route. There are uncertainties regarding other possible highly exposed sub-populations, *e.g.*, swimmers, those who use humidifiers, hot-tubs, and outdoor misters, persons living near industrial sources, people working in laundromats, and persons working with pesticides employing chloroform as a solvent (USEPA 1998b).

A 1998 International Life Sciences Institute (ILSI) report evaluated the uptake of drinking water contaminants through the skin and by inhalation. The report noted that ''(i)n the case of chloroform, its high volatility leads to its rapid movement from liquid to air. Large water-use sources, such as showers, become dominant sources with respect to exposure'' and ''(t)he inhalation route is demonstrated to be the primary route for higher-volatility compounds (*e.g.,* chloroform)'' (ILSI 1998). Weisel and Jo (1996) found that ''approximately equivalent amounts of chloroform from water can enter the body by three different exposure routes, inhalation, dermal absorption, and ingestion, for typical daily activities of drinking and bathing.''

Chloroform has been found in beverages, especially soft drinks, and food, particularly dairy products (Wallace, 1997). Wallace states that ''ingestion (drinking tap water and soft drinks and eating certain dairy foods), inhalation (breathing peak amounts of chloroform emitted during showers or baths, and lower levels in indoor air from other indoor sources), and dermal absorption (during showers, baths, and swimming)'' each ''appear to be potentially substantial contributors to total exposure''.

EPA estimates that for the median individual, ingestion of total tap water (assuming certain activity patterns, habits, and home characteristics) can contribute roughly 28 percent of the total dose of chloroform (USEPA 2001a). With assumptions as described, tap water ingestion is a portion of exposure through fluid intake which contributes about 34 percent of the total dose, inhalation accounts for about 31 percent of the total dose, ingestion of foods contributes another 27 percent of the overall dose, and dermal absorption (primarily during showering) adds slightly less than 8 percent of the total dose. These exposure percentages are based on average daily doses (mean chloroform intake for adults) for each source and route of exposure under specific conditions. They do not take into account the considerable variability in several factors across the population. For instance, intake of drinking water or particular foods and length of shower varies from day-to-day, as do home air turnover rates and ventilation. Different areas in the United States vary with respect to these factors and chloroform concentrations in food. Thus, although the 28 percent for the median individual is based on reasonable assumptions, uncertainty remains.

Given the uncertainties of estimation, EPA believes available analyses point to the RSC of 20 percent as the appropriate default (*i.e.,* 20 percent of exposure to chloroform comes from drinking tap water alone). EPA also believes that this default is protective of public health and is a more reasonable choice than choosing any particular estimate because of the assumptions and uncertainties involved with each estimation. Hence, EPA is proposing the MCLG based on the RSC default of 20 percent which supports the adequacy of the margin of safety associated with the MCLG.

iii. *Water ingestion and body weight assumptions.* In MCLG calculations, EPA assumes the 90th percentile water ingestion of 2 liters (roughly equivalent to a half gallon) per day (USEPA 2000a). The use of a conservative consumption estimate is consistent with the objective of setting an MCLG that is protective. EPA also uses a default adult body weight of 70 kg (equal to 154 pounds) for the RfD since dose is calculated from lifetime studies of animals and compared to lifetime exposure for humans.

iv. *MCLG calculation.* The MCLG is calculated to be 0.07 mg/L using the following assumptions: an adult tap water consumption of 2 L per day for a 70 kg adult, and a relative source contribution of 20%:

$$\text{MCLG for Chloroform} = \frac{0.01\ \text{mg/kg/d} \times 70\ \text{kg} \times 0.2}{2\text{L/day}} = 0.07\ \text{mg/L (rounded)}$$

EPA concludes that an MCLG of 0.07 mg/L based on protection against liver toxicity will be protective against carcinogenicity given that the mode of action for chloroform involves cytotoxicity as a key event preceding tumor development. Therefore, the recommended MCLG for chloroform is 0.07 mg/L.

v. *Other considerations.* The evidence supports similarity of potential response in children and adults. The basic biology of toxicity caused by cell damage due to oxidative damage is expected to be the same. There is nothing about the incidence and etiology of liver and kidney cancer in children to indicate that they would be inherently more sensitive to this mode of action. Most importantly in this case, children appear to be no different quantitatively in ability to carry out the oxidative metabolism step for the induction of toxicity and cancer and may, as fetuses, be less susceptible (USEPA 1999c).

Some commenters on the March 1998 NODA were concerned that EPA did not take drinking water epidemiology studies into account in its evaluation of chloroform risk. EPA believes that while the epidemiologic evidence suggests that chlorinated drinking water may be associated with certain cancers and reproductive, developmental effects pertinent to the risk of disinfectant byproduct mixtures, it does not provide insight into the risk from chloroform specifically. The SAB noted that ''(t)he goal of the draft risk assessment (the isolation of the effect of chloroform in drinking water) makes the extensive epidemiologic evidence on drinking water disinfection byproducts largely irrelevant'' to the specific question of chloroform health risks because, in the available studies, chloroform cannot be isolated from other disinfection byproducts that may be in the drinking water (USEPA 2000f). The SAB noted that ''the epidemiologic evidence is quite pertinent to the broader question of most direct regulatory concern, namely disinfection byproducts in the aggregate''.

d. *Feasibility of other options.* During the development of the MCLG for chloroform, EPA considered a number of options for both the chloroform MCLG and the TTHM MCL. Today, EPA is proposing the preferred option of a 0.07 mg/L MCLG for chloroform. EPA primarily considered two other options which are discussed in more detail later: a 0.07 mg/L MCLG for chloroform in conjunction with developing MCLs for each of the individual TTHMs (*i.e.,* 4 MCLs and 4 MCLGs for the THMs); and developing a single combined MCLG for TTHM rather than developing a separate MCLG for each of the THMs.

EPA considered developing separate MCLGs and MCLs for each THM. Under this strategy, EPA would determine an MCL as close to the individual MCLGs as is technically feasible, taking cost into consideration, for each THM. EPA would propose an MCLG of 0.07 mg/L for chloroform and maintain the Stage 1 DBPR MCLGs for BDCM, DBCM, and bromoform (USEPA 1998c). EPA analyzed the impact such an MCL strategy would have and ultimately rejected this option. This approach represents a fundamental shift from the TTHM strategy agreed to by stakeholders and EPA as part of the M–DBP negotiation process and reflected in the 1998 Stage 1 DBPR. In addition, one important component of the existing single MCL is that TTHMs are an indicator for other DBPs. Developing a separate MCL for each THM would move away from this indicator approach. Because precursor and DBP occurrence measurements are highly variable, both temporally and geographically, determining technical feasibility for best available technology (BAT) would be difficult. Compliance with individual THM standards would be very different from compliance based on a sum of the four THMs and it is not clear what treatment technology shifts would be needed. This problem would be particularly exacerbated in areas with high bromide, such as California. EPA also projected that States would have a difficult time overseeing (*e.g.*, variances, exemptions, *etc.*) the more complicated rule that would result from this option.

EPA considered establishing a single combined MCLG for TTHM. There is precedent for using a toxicity equivalency quotient (analogous to a combined MCLG) for dioxin and coplanar PCBs (USEPA 2000o, Draft Dioxin Reassessment). From a scientific standpoint, a combined MCLG approach requires that the chemicals have a similar mode of action and health endpoint. Chemicals within each of the dioxin and coplanar PCB classes have the same mode of action and endpoint (target tissue). Within the PCB class, noncoplanar PCBs have a different mode of action than the coplanar PCBs. Noncoplanar PCBs are, therefore, not included in the toxicity equivalency quotient for coplanar PCBs. In the case of the disinfection byproducts, EPA believes that the THMs have different modes of action and health endpoints. One of the THMs is a liver carcinogen (chloroform) with a mode of action dependent on cytolethality; two are DNA-reactive carcinogens (bromodichloromethane—large intestine and kidney tumors, and bromoform—large intestine tumors); and one is a nonlinear non-carcinogen (dibromochloromethane) which is a liver toxicant. EPA therefore, chose not to develop a combined MCLG for TTHM. Consequently, after considering this alternative option in some detail, EPA is today proposing an MCLG of 0.07 mg/L for chloroform.

3. Request for Comment

Based on the information presented previously, EPA is proposing an MCLG for chloroform of 0.07 mg/L. EPA requests comments on the MCLG and on EPA's cancer assessment for chloroform. EPA also requests comments on the RfD, the default RSC of 20 percent, and the tap water consumption and body weight assumptions used in the MCLG calculation. EPA solicits additional data on chloroform exposure via other sources and routes. EPA requests comment on the other options for developing the chloroform MCLG that the Agency considered.

B. MCLGs for THMs and HAAs

1. What Is EPA Proposing Today?

Today EPA is proposing new MCLGs of 0.02 mg/L for TCAA and 0.03 mg/L for MCAA based on new toxicological data. As a part of the Stage 1 DBPR, EPA finalized an MCLG of 0.3 mg/L for TCAA. The Stage 1 DBPR did not include an MCLG for MCAA (although it was included as one of the five haloacetic acids in the HAA5 MCL). With the exception of chloroform, discussed above, and these two HAAs, EPA is not revising any of the other MCLGs that were finalized in the Stage 1 DBPR. No significant new studies that would change EPA's MCLG estimates for BDCM, DBCM, bromoform, or DCAA have been published since the Stage 1 DBPR. See section III for a summary of new health effects data.

2. How Was This Proposal Developed?

EPA reviewed the available literature on BDCM, DBCM, bromoform, DCAA and determined that there was no new

information that would cause EPA to revise its MCLG estimates. New toxicology studies on reproductive and developmental effects and cancer are summarized in sections III.B. and III.D. of today's proposal.

EPA is proposing new MCLGs for TCAA and MCAA. The health effects information and studies described in the following two sections that support the proposed MCLGs are summarized from the Addendum to the Criteria Document for Monochloroacetic Acid and Trichloroacetic Acid (USEPA 2003b). The occurrence of MCAA and TCAA are discussed in the *Stage 2 Occurrence Assessment for Disinfectants and Disinfection Byproducts* (USEPA 2003o). a. Trichloroacetic acid. In the final Stage 1 DBPR, EPA based its health effects assessment of TCAA on developmental toxicity and limited evidence of carcinogenicity (USEPA 1998c). Since then, the Agency has decided that the RfD based on a developmental LOAEL yields a less conservative RfD than that based on liver toxicity derived from the study by DeAngelo *et al.* (1997). Thus, the Agency has reassessed the health effects of TCAA based on liver toxicity and revised the RfD and MCLG.

TCAA induces systemic, noncancer effects in animals and humans that can be grouped into three categories: metabolic alterations, liver toxicity; and developmental toxicity. The primary site of TCAA toxicity is the liver (USEPA1994a; Dees and Travis, 1994; Acharya *et al.* 1995; Acharya *et al.* 1997; DeAngelo *et al.*1997).

The liver has consistently been identified as a target organ for TCAA toxicity in short-term (Goldsworthy and Popp, 1987; DeAngelo *et al.* 1989; Sanchez and Bull, 1990) and longer-term (Bull *et al.* 1990; Mather *et al.* 1990; Bhat *et al.* 1991) studies. Peroxisome proliferation has been a primary endpoint evaluated, with mice reported to be more sensitive to this effect than rats. More recent studies have confirmed these earlier findings. TCAA-induced peroxisome proliferation was observed in B6C3F1 mice exposed for 10 weeks to doses as low as 25 mg/kg/day (Parrish *et al.* 1996), while in rats exposed to TCAA for up to 104 weeks (DeAngelo *et al.* 1997), peroxisome proliferation was observed at 364 mg/kg/day, but not at 32.5 mg/kg/day. Increased liver weight and significant increases in hepatocyte proliferation have been observed in short-term studies in mice at doses as low as 100 mg/kg/day (Dees and Travis, 1994), but no increase in hepatocyte proliferation was noted in rats given TCAA at similar doses (DeAngelo *et al.* 1997). More clearly adverse liver toxicity endpoints, including increased serum levels of liver enzymes (indicating leakage from cells) or histopathological evidence of necrosis, have been reported in rats, but generally only at high doses. For example, in a rat chronic drinking water study, increased hepatocyte necrosis was observed at a dose of 364 mg/kg/day (DeAngelo *et al.* 1997).

In the DeAngelo *et al.*(1997) study, groups of 50 male F344 rats were administered TCAA in drinking water, at 0, 50, 500, or 5000 mg/L, resulting in time-weighted mean daily doses of 0, 3.6, 32.5, or 364 mg/kg for 104 weeks. There were no significant differences in water consumption or survival between the control and treatment groups. Exposure to the high dose of TCAA resulted in a significant decrease in body weight of 11% at the end of the study. The absolute but not relative liver weight was decreased at the high dose. Complete necropsy and histopathology examination showed mild hepatic cytoplasmic vacuolization in the two low-dose groups, but not in the high-dose group. The severity of hepatic necrosis was increased mildly in the high-dose animals. Analyses of serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities at the end of exposure showed a significant decrease in AST activity in the mid-dose group and a significant increase in ALT level in the high-dose group. Since increased serum ALT or AST levels reflect hepatocellular necrosis, the increased ALT at the high dose is considered an adverse effect, while a non-dose related decrease of AST is not. Peroxisome proliferation was increased significantly in the high-dose animals. There was no evidence of any exposure-related increase in hepatocyte proliferation. Based on the significant decrease in body weight (≥10%), minimal histopathology changes, and increased serum ALT level, the high dose of 364 mg/kg/day is considered the LOAEL and the mid dose of 32.5 mg/kg/day is considered the NOAEL.

There are no reproductive toxicity studies of TCAA. The results of an *in vitro* fertilization assay indicated that TCAA might decrease fertilization (Cosby and Dukelow, 1992). The available data suggest that TCAA is a developmental toxicant. TCAA increased resorptions, decreased implantations, and increased fetal cardiovascular malformations when administered to pregnant rats at 291 mg/kg/day (Johnson *et al.* 1998) on gestation days 1–22. In another study, decreased fetal weight and length, and increased cardiovascular malformations were observed when pregnant rats were administered 330 mg/kg/day TCAA by gavage during gestation days 6 to 15 (Smith *et al.* 1989). Neither of these studies identified a NOAEL. The results of *in vitro* developmental toxicity assays, including mouse and rat whole-embryo culture (Saillenfait *et al.* 1995; Hunter *et al.* 1996) and frog embryo teratogenesis assay—*Xenopus* (FETAX) (Fort *et al.* 1993) yielded positive results. The *Hydra* test system (Fu *et al.* 1990) produced negative results.

TCAA has been reported to induce liver tumors in mice but not in rats (USEPA 1994a). This observation has also been made in more recent drinking water studies. Pereira (1996) observed an increased incidence of hepatocellular adenomas and carcinomas in female B6C3F1 mice at doses of 262 mg/kg/day and higher after 82 weeks. In contrast, no increase in neoplastic liver lesions were found in F344 rats given doses up to 364 mg/kg/day for 104 weeks (DeAngelo *et al.* 1997). In addition, a variety of recent mechanistic studies have observed that TCAA either induced or promoted liver tumors in mice (Ferreira-Gonzalez *et al.* 1995; Pereira and Phelps, 1996; Tao *et al.* 1996; Latendresse and Pereira, 1997; Stauber and Bull, 1997; Tao *et al.* 1998).

Recent mutagenicity data have provided mixed results (Giller *et al.* 1997; DeMarini *et al.* 1994; Harrington-Brock *et al.* 1998). TCAA did not induce oxidative DNA damage in mice following dosing for either 3 or 10 weeks (Parrish *et al.* 1996). Studies on DNA strand breaks and chromosome damage produced mixed results (Nelson and Bull, 1988; Chang *et al.* 1991; Mackay *et al.* 1995; Harrington-Brock *et al.* 1998).

According to the 1999 Draft Guidelines for Carcinogen Risk Assessment (USEPA 1999a), a compound is appropriately classified as ''Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential'' when ''the evidence from human or animal data is suggestive of carcinogenicity, which raises a concern for carcinogenic effects but is judged not sufficient for a conclusion as to human carcinogenic potential''. Based on uncertainty surrounding the relevance of the liver tumor data in B6C3F1 mice, TCAA can best be described as ''Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential'' under the 1999 Draft Guidelines for Carcinogen Risk Assessment. Thus a quantitative estimate of cancer potency is not supported.

The RfD for TCAA of 0.03 mg/kg/day is based on the NOAEL of 32.5 mg/kg/day for liver histopathological changes identified by DeAngelo *et al.* (1997). The RfD includes an uncertainty factor of 1000 (composite uncertainty factor consisting of three factors of 10 chosen to account for extrapolation from a NOAEL in animals, inter-individual variability in humans, and insufficiencies in the database, including the lack of full histopathological data in a second species, the lack of a developmental toxicity study in second species, and the lack of a multi-generation reproductive study).

The MCLG is calculated to be 0.02 mg/L using the following assumptions: an adult tap water consumption of 2 L of tap water per day for a 70 kg adult, a relative source contribution (RSC) of 20%, and an additional safety factor to account for possible carcinogenicity. EPA has traditionally applied an additional safety factor of 1–10 beyond the uncertainty factors included in the RfD to the MCLG to account for possible carcinogenicity in cases where there is limited evidence of carcinogenicity from drinking water, considering weight of evidence, pharmacokinetics, potency and exposure (USEPA 1994b, p.38678). EPA is proposing this additional safety factor of 10 for TCAA for the following reasons: TCAA causes liver tumors in mice but does not do so in rats. In addition, although peroxisome proliferation (a mode of action of limited relevance to humans) may play a role in the development of the mouse tumors, rats also exhibit a peroxisomal proliferative response after exposure to TCA, yet do not develop tumors. Other data suggest that promotion of initiated cells and/or disrupted cell signaling may be involved in the mode of action for the mouse tumors. Together these factors argue against quantification of the mouse liver tumors using linear extrapolation from the dose-response curve, but are not sufficient to rule out concern for a tumorigenic response. Accordingly, EPA has employed the ten-fold additional safety factor in determination of the Lifetime Health Advisory for TCAA. EPA requests comment on the use of 10 as the additional safety factor for possible carcinogenicity.

$$\text{MCLG for TCAA} = \frac{(0.03\ \text{mg/kg/day})(70\ \text{kg})(20\%)}{(2\ \text{L/day})(10)} = 0.02\ \text{mg/L (rounded)}$$

An RSC factor of 20% is used to account for exposure to TCAA in sources other than tap water, such as ambient air and food. Although TCAA is nonvolatile and inhalation while showering is not expected to be a major contribution to total dose, rain waters contain 0.01–1.0 μg/L of TCAA (Reimann *et al.* 1996) and it can be assumed to be detected in the atmosphere. Limited data on concentrations of TCAA in air (NATICH 1993) indicate inhalation of TCAA in ambient air may contribute to overall exposure. Concentrations of TCAA that have been measured in a limited selection of foods including vegetables, fruits, grain and bread (Reimann *et al.* 1996) are comparable to that in water. About 3 to 33% of TCAA in cooking water have been reported to be taken up by the food during cooking in a recent research summary (Raymer *et al.* 2001). In addition, there are uses of chlorine in food production and processing, and TCAA may occur in food as a byproduct of chlorination (USEPA 1994a). Therefore, ingestion of TCAA in food may also contribute to the overall exposure. A recent dermal absorption study of DCAA and TCAA from chlorinated water suggested that the dermal contribution to the total doses of DCAA and TCAA from routine household uses of drinking water is less than 1% (Kim and Weisel, 1998).

b. *Monochloroacetic acid.* Subchronic and chronic oral dosing studies suggest that the primary targets for MCAA-induced toxicity include the heart and nasal epithelium. In a 13-week oral gavage study, decreased heart weight was observed at 30 mg/kg/day and cardiac lesions progressed in severity with increasing dose. Liver and kidney toxicity were only observed at higher doses (NTP 1992). In a two-year study, decreased survival and nasal and forestomach hyperplasia were observed in mice at 50 mg/kg/day (NTP 1992). A more recent study confirms the heart and nasal cavities as target sites for MCAA. DeAngelo *et al.* (1997) noted decreased body weight at 26.1 mg/kg/day and myocardial degeneration and inflammation of the nasal cavities in rats exposed to doses of 59.9 mg/kg/day for up to 104 weeks.

No studies were located on the reproductive toxicity of MCAA and the potential developmental toxicity of MCAA has not been adequately tested. Two developmental toxicity studies were identified. Johnson *et al.* (1998) reported markedly decreased maternal weight gain, but no developmental effects, in rats exposed to 193 mg/kg/day MCAA through gestation days 1–22, only fetal heart was examined. In contrast, in a published abstract, Smith *et al.* (1990) reported an increase in cardiovascular malformations when pregnant rats were exposed to 140 mg/kg/day; this was also the LOAEL for maternal toxicity, based on marked decreases in weight gain. MCAA was noted as a potential developmental toxicant in *in vitro* screening assays using *Hydra* (Fu *et al.* 1990; Ji *et al.* 1998).

MCAA has yielded mixed results in genotoxicity assays (USEPA 1994a; Giller *et al.* 1997), but has not induced a carcinogenic response in chronic rodent bioassays (NTP 1992; DeAngelo *et al.* 1997). In chronic oral gavage studies, a LOAEL of 15 mg/kg/day (the lowest dose tested) for decreased survival was identified in rats. In mice the NOAEL was 50 mg/kg/day and the LOAEL was 100 mg/kg/day for nasal and forestomach epithelium hyperplasia (NTP 1992). In a more recent chronic study, DeAngelo *et al.* (1997) reported a LOAEL of 3.5 mg/kg/day in rats given MCAA in their drinking water, based on increased absolute and relative spleen weight. Although spleen weight was decreased at the mid and high doses, this might reflect the masking effect of overt toxicity. As evidence for this, decreased body weight (>10%), liver, kidney, and testes weight changes were reported beginning at the next higher dose of 26.1 mg/kg/day. No increased spleen weight was reported in the NTP (1992) bioassays, but the lowest dose in rats caused severe toxicity, and the lowest dose in mice was more than an order of magnitude higher than the LOAEL in the DeAngelo *et al.* (1997) study.

According to the 1999 Draft Guidelines for Carcinogen Risk Assessment (USEPA 1999a), a compound is appropriately classified as ''Not Likely to be Carcinogenic to Humans'' when it has ''been evaluated in at least two well-conducted studies in two appropriate animal species without demonstrating carcinogenic effects.'' MCAA can best be described as ''Not Likely to be Carcinogenic to Humans'' under the 1999 Draft Guidelines for Carcinogen Risk Assessment.

The RfD for MCAA of 0.004 mg/kg/day is based on a LOAEL of 3.5 mg/kg/day for increased spleen weight in rats (DeAngelo *et al.* 1997) and application of an uncertainty factor of 1000 (composite uncertainty factor consisting of two factors of 10 chosen to account for extrapolation from an animal study, and inter-individual variability in humans; as well as two factors of 3 for extrapolation from a minimal effect LOAEL, and insufficiencies in the database, including the lack of adequate developmental toxicity studies in two species, and the lack of a multi-generation reproductive study). Two developmental toxicity studies have been reported (Johnson *et al.* 1998; Smith *et al.* 1990), but the NOAELs yielded less conservative RfDs. The study by DeAngelo et al (1997) is the most appropriate for derivation of the RfD because it identifies the lowest LOAEL, and dosing was in drinking water, which is more appropriate for human health risk assessment.

The MCLG is calculated to be 0.03 mg/L using the following assumptions: an adult tap water consumption of 2 L of tap water per day for a 70 kg adult, and a relative source contribution of 20 %.

$$\text{MCLG for MCAA} = \frac{(0.004\ \text{mg/kg/day})(70\ \text{kg})(20\%)}{(2\ \text{L/day})} = 0.03\ \text{mg/L (rounded)}$$

An RSC factor of 20% is used to account for exposure to MCAA in other sources in addition to tap water. Although MCAA is nonvolatile and inhalation while showering is not expected to be a major contribution to total dose, rain waters contain 0.05–9 µg/L of MCAA (Reimann *et al.* 1996) and it can be assumed to be detected in the atmosphere. Presence of MCAA has also been reported in rain waters; thus, inhalation of MCAA in ambient air may contribute to overall exposure. Concentrations of MCAA that have been measured in a limited selection of foods including vegetables, fruits, grain and bread (Reimann *et al.* 1996) are comparable to that in water. About 2.5 to 62% of MCAA in cooking water has been reported to be taken up by food during cooking in a recent research summary (Raymer *et al.* 2001). In addition, there are uses of chlorine in food production and processing, and MCAA may occur in food as a byproduct of chlorination (USEPA 1994a). Therefore, ingestion of MCAA in food may also contribute to the overall exposure. Assuming dermal absorption rate of MCAA is similar to DCAA, dermal contribution to the total doses of MCAA from routine household uses of drinking water should be minor (*see* V.B.2.a.).

3. Request for Comment

EPA requests comment on the new MCLGs for TCAA (0.02 mg/L) and MCAA (0.03 mg/L) and all the factors incorporated in the derivation of the MCLGs, including the RfDs and RSCs. EPA also solicits health effect information on DBAA and monobromoacetic acid (MBAA), for which MCLGs have not yet been established.

C. Consecutive Systems

Today's proposal includes provisions for consecutive systems, which are public water systems that purchase or otherwise receive finished water from another water system (a wholesale system). As described in this section, consecutive systems face particular challenges in providing water that meets regulatory standards for DBPs and other contaminants whose concentration can increase in the distribution system. Moreover, current regulation of DBP levels in consecutive systems varies widely among States. In consideration of these factors, EPA is proposing monitoring, compliance schedule, and other requirements specifically for consecutive systems. These requirements are intended to facilitate compliance by consecutive systems with MCLs for TTHM and HAA5 under the Stage 2 DBPR. Further, this approach will help to ensure that consumers in consecutive systems receive equivalent public health protection. This section begins with a summary of how EPA proposes to regulate consecutive systems under the Stage 2 DBPR. The intent of this section is to provide an overview of all consecutive system requirements in today's proposal. Detailed explanations of these requirements are provided in later sections of this preamble. The overview of consecutive system requirements is followed by an explanation of why EPA has taken this approach to consecutive systems in today's proposal, including recommendations from the Stage 2 M–DBP Federal Advisory Committee.

1. What Is EPA Proposing Today?

As public water systems, consecutive systems must provide water that meets the MCLs for TTHM and HAA5 under the proposed Stage 2 DBPR, and must carry out associated monitoring, reporting, recordkeeping, public notification, and other requirements. The following discussion summarizes how the Stage 2 DBPR requirements apply to consecutive systems, beginning with a series of definitions. Later sections of this preamble provide further details as noted.

a. *Definitions.* To address consecutive systems in the Stage 2 DBPR, the Agency must define them, along with a number of related terms.

EPA is proposing to define a *consecutive system* in the Stage 2 DBPR as a public water system that buys or otherwise receives some or all of its finished water from one or more wholesale systems for at least 60 days per year. In addition to buying finished water, some consecutive systems also operate a treatment plant (meaning a plant that treats source water to produce finished water). As described in section V.I., monitoring requirements under the Stage 2 DBPR proposal differ depending on whether a consecutive system buys all of its finished water year-round or, alternatively, produces some of its finished water through treating source water.

EPA proposes to define finished water as water that has been introduced into the distribution system of a public water system and is intended for distribution without further treatment, except that necessary to maintain water quality (such as booster disinfection). With this definition, water entering the distribution system is finished water even if a system subsequently applies additional treatment like booster disinfection to maintain a disinfectant residual throughout the distribution system.

In today's proposal, EPA defines a *wholesale system* as a public water system that treats source water and then sells or otherwise delivers finished water to another public water system for at least 60 days per year. Delivery may be through a direct connection or through the distribution system of another consecutive system. Under this definition, a consecutive system that passes water from a wholesaler to another consecutive system, and that does not also treat source water, is not

a wholesale system. Rather, the system that actually produces the finished water is responsible for wholesale system requirements under the proposed Stage 2 DBPR.

A *consecutive system entry point* is defined as a location at which finished water is delivered at least 60 days per year from a wholesale system to a *consecutive system*. Section V.I. presents the relationship between consecutive system entry points and proposed Stage 2 DBPR monitoring requirements. The *combined distribution system* is the interconnected distribution system consisting of the distribution systems of wholesale systems and of the consecutive systems that receive finished water from those wholesale system(s).

b. *Monitoring.* For consecutive systems that both purchase finished water and treat source water to produce finished water for at least part of the year, EPA is proposing monitoring requirements under a treatment plant-based approach, described in section V.I. This is the approach proposed for non-consecutive systems under the Stage 2 DBPR as well. Under this approach, the sampling requirements for consecutive systems will be influenced by both the number of treatment plants operated by the system and the number of consecutive system entry points, as well as population served and source water type.

For consecutive systems that purchase all of their finished water year-round, EPA is proposing monitoring requirements under a population-based approach, also described in section V.I. Under the population-based approach, the population of the consecutive system will determine the sampling requirements. EPA believes this approach is more appropriate than plant-based monitoring because these consecutive systems do not have treatment plants. As noted in section V.I., EPA is requesting comment on extending population-based monitoring to all systems, including non-consecutive systems. EPA has prepared draft guidance for implementing the IDSE monitoring requirements (described in section V.H.) using the population-based approach (USEPA 2003j).

EPA is also proposing that States have the opportunity to specify alternative monitoring requirements for multiple consecutive systems in a combined distribution system. This option allows States to consider complex consecutive system configurations for which alternative monitoring strategies might be more appropriate. As a minimum under such an approach, each consecutive system must collect at least one sample among the total number of samples required for the combined distribution system and will base compliance on samples collected within its distribution system. The consecutive system is responsible for ensuring that required monitoring is completed and the system is in compliance. The consecutive system may conduct the monitoring itself or arrange for the monitoring to be done by the wholesale system or another outside party. Whatever approach it chooses, the consecutive system must document its monitoring strategy as part of its DBP monitoring plan.

Finally, EPA is proposing that consecutive systems not conducting disinfectant residual monitoring comply with the monitoring requirements and MRDLs for chlorine and chloramines.

c. Compliance schedules. EPA is proposing that consecutive systems of any size comply with the requirements of the Stage 2 DBPR on the same schedule as required for the largest system in the combined distribution system. This includes the schedule for carrying out the IDSE, described in section V.H, and for meeting the Stage 2B MCLs for TTHM and HAA5, described in section V.D. As discussed later in this section, EPA is proposing simultaneous compliance schedules under the Stage 2 DBPR for all systems (both wholesalers and consecutive systems) in a combined distribution system because this may allow for more cost-effective compliance with TTHM and HAA5 MCLs. This is also consistent with the recommendations of the Stage 2 M–DBP Advisory Committee. See section V.J for details of compliance schedule requirements.

d. *Treatment.* While consecutive systems often do not need to treat finished water received from a wholesale system, they may need to implement procedures to control the formation of DBPs in the distribution system. For consecutive systems, EPA is proposing that the BAT for meeting TTHM and HAA5 MCLs is chloramination with management of hydraulic flow and storage to minimize residence time in the distribution system. This BAT stems from the recognition that treatment to remove already-formed DBPs or minimize further formation is different from treatment to prevent or reduce their formation. See section V.F for additional information on BATs and their role in compliance with MCLs.

e. *Violations.* Under this proposal, monitoring and MCL violations are assigned to the PWS where the violation occurred. Several examples are as follows:

—If a consecutive system has hired its wholesale system under contract to monitor in the consecutive system and the wholesale system fails to monitor, the consecutive system is in violation because it has the legal responsibility for monitoring under State/EPA regulations.

—If monitoring results in a consecutive system indicate an MCL violation, the consecutive systems is in violation because it has the legal responsibility for complying with the MCL under State/EPA regulations. The consecutive system may set up a contract with its wholesale system that details water quality delivery specifications.

—If a wholesale system has a violation and provides that water to a consecutive system, the wholesale system is in violation. Whether the consecutive system is in violation will depend on the situation. The consecutive system will also be in violation unless it conducted monitoring that showed that the violation was not present in the consecutive system.

f. *Public notice and consumer confidence reports.* The responsibilities for public notification and consumer confidence reports rest with the individual system. Under the Public Notice Rule and Consumer Confidence Report Rule, the wholesale system is responsible for notifying the consecutive system of analytical results and violations related to monitoring conducted by the wholesale system. Consecutive systems are required to conduct appropriate public notification after a violation (whether in the wholesale system or the consecutive system). In their consumer confidence report, consecutive systems must include results of the testing conducted by the wholesale system unless the consecutive system conducted equivalent testing that indicated the consecutive system was in compliance, in which case the consecutive system reports its own compliance monitoring results.

g. *Recordkeeping and reporting.* Consecutive systems are required to keep all records required of PWSs regulated under this rule. They are also required to report to the State monitoring results, violations, and other actions, and are required to consult with the State after a significant excursion.

h. *State special primacy conditions.* EPA is aware that due to the complicated wholesale system-consecutive system relationships that

exist nationally, there will be cases where the standard monitoring framework proposed today will be difficult to implement. Therefore, the Agency is proposing to allow States to develop, as a special primacy condition, a program under which the State can modify monitoring requirements for consecutive systems. These modifications must not undermine public health protection and all systems, including consecutive systems, must comply with the TTHM and HAA5 MCLs based on the LRAA. However, such a program would allow the State to establish monitoring requirements that account for complicated distribution system relationships, such as where neighboring systems buy from and sell to each other regularly throughout the year, water passes through multiple consecutive systems before it reaches a user, or a large group of interconnected systems have a complicated combined distribution system. EPA intends to develop a guidance manual to address development of a State program and other consecutive system issues.

2. How Was This Proposal Developed?

The practice of public water systems buying and selling water to each other has been commonplace for many years. Reasons include saving money on pumping, treatment, equipment, and personnel; assuring an adequate supply during peak demand periods; acquiring emergency supplies; selling surplus supplies; delivering a better product to consumers; and meeting Federal and State water quality standards. EPA estimates that there are at least 8500 consecutive systems nationally, based on the definitions being proposed today.

Consecutive systems face particular challenges in providing water that meets regulatory standards for contaminants that can increase in the distribution system. Examples of such contaminants include coliforms, which can grow if favorable conditions exist, and some DBPs, including THMs and HAAs, which can increase when a disinfectant and DBP precursors continue to react in the distribution system.

EPA is proposing requirements specifically for consecutive systems because States have taken widely varying approaches to regulating DBPs in consecutive systems. For example, some States do not regulate DBP levels in consecutive systems that deliver disinfected water but do not add a disinfectant. Other States determine compliance with DBP standards based on the combined distribution system that includes both the wholesaler and consecutive systems. In this case, sites in consecutive systems are treated as monitoring sites within the combined distribution system. Once fully implemented, this proposed rule will ensure similar protection for consumers in consecutive systems.

EPA is proposing that consecutive systems and wholesale systems be on the same compliance schedule because generally the most cost-effective way to achieve compliance with TTHM and HAA5 MCLs is to treat at the source, typically through precursor removal or alternative disinfectants. For a wholesale system to make the best decisions concerning the treatment steps necessary to meet TTHM and HAA5 LRAAs under the Stage 2 DBPR, both in its own distribution system and in the distribution systems of consecutive systems it serves, the wholesale system must know the DBP levels throughout the combined distribution system. Without this information, the wholesale system may design treatment changes that allow the wholesale system to achieve compliance, but leave the consecutive system out of compliance. EPA also recognizes that there may be cases where a consecutive system needs to add treatment even after a wholesale system has optimized its own treatment train.

In consideration of these issues, the Stage 2 M–DBP Advisory Committee recognized two principles related to consecutive systems: (1) Consumers in consecutive systems should be just as well protected as customers of all systems, and (2) monitoring provisions should be tailored to meet the first principle. Accordingly, the Advisory Committee recommended that all wholesale and consecutive systems comply with provisions of the Stage 2 DBPR on the same schedule required of the wholesale or consecutive system serving the largest population in the combined distribution system. In addition, the Advisory Committee recommended that EPA solicit comments on issues related to consecutive systems that the Advisory Committee had not fully explored (USEPA 2000g). EPA agrees with these recommendations and they are reflected in today's proposal.

3. Request for Comment

EPA requests comment on all consecutive system issues related to this rule. Specifically, EPA requests comment on the following:

—Whether the proposed definitions adequately address various wholesale system-consecutive system relationships and issues.

—Whether any additional terms need to be defined and, if so, what the definition should be.

—Whether the criteria for States' use of the special primacy criteria and other State responsibilities are appropriate and adequate.

—Whether it is necessary to require that consecutive system treatment be installed on the same compliance schedule as the wholesale system in cases where the size of the consecutive system might otherwise allow it a longer compliance time frame and the consecutive system treatment does not affect water quality in any other system.

D. MCLs for TTHM and HAA5

1. What Is EPA Proposing Today?

Today, EPA is proposing use of locational running annual averages (LRAAs) to determine compliance with the MCLs for TTHM and HAA5. Consistent with the Stage 2 M-DBP Advisory Committee recommendation, EPA is proposing a phased approach for LRAA implementation to allow systems to identify compliance monitoring locations for Stage 2B while facilitating transition to the new compliance strategy and maintaining simultaneous compliance schedules for the Stage 2 DBPR and the LT2ESWTR.

In Stage 2A, all systems must comply with MCLs of 0.120 mg/L for TTHM and 0.100 mg/L for HAA5 as LRAAs using Stage 1 DBPR compliance monitoring sites. In addition, during this time period, all systems must continue to comply with the Stage 1 DBPR MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as RAAs.

In Stage 2B, all systems, including consecutive systems, must comply with MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as LRAAs using sampling sites identified under the Initial Distribution System Evaluation (IDSE) (discussed in section V.H.).

Details of proposed monitoring requirements and compliance schedules are discussed in preamble sections V.I. and V.J., respectively, and may be found in § 141.136 and subpart V of today's rule.

2. How Was This Proposal Developed?

a. *Definition of an LRAA.* The primary objective of the LRAA is to reduce exposure to high DBP levels. For an LRAA, an annual average must be computed at each monitoring site. The RAA compliance basis of the 1979 TTHM rule and the Stage 1 DBPR allows a system-wide annual average under which high DBP concentrations in one or more locations are averaged with, and

dampened by, lower concentrations elsewhere in the distribution system. Figure V–1 illustrates the difference in calculating compliance with the MCLs for TTHM between a Stage 1 DBPR RAA, and the proposed Stage 2 DBPR LRAA.

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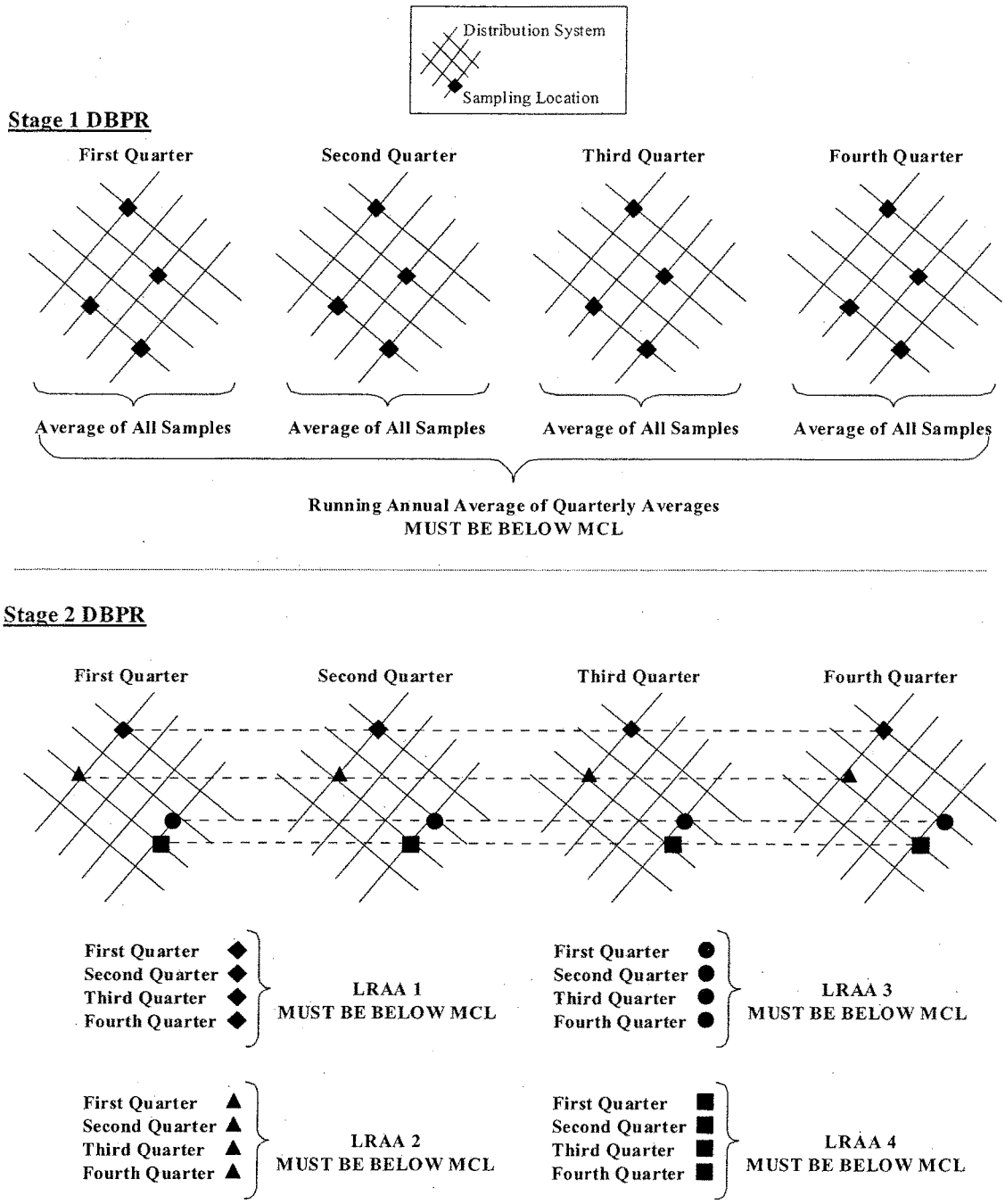
Figure V-1. Comparison of RAA and LRAA compliance calculations[1].



[1]Stage 2 DBPR sampling locations will be selected based on the results of an IDSE study and may occur at locations different from Stage 1 DBPR sampling sites.

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b. *Consideration of regulatory alternatives.* This section will discuss EPA's and the Stage 2 M-DBP Advisory Committee's decision-making process as an array of alternative MCL strategies were considered. EPA believes that the MCL alternative proposed today (MCLs of 0.080 mg/L TTHM, 0.060 mg/L HAA5 as LRAAs) is supported by the best available research, data, and analysis. The science related to cancer and reproductive and developmental health effects that may be associated with DBPs, in conjunction with occurrence data that show that a significant number of high DBP levels occur under current regulatory scenarios, justify a change in regulation. EPA believes that this proposal achieves an appropriate balance between the available science and the uncertainties. EPA believes that regulatory action is necessary and prudent in the interest of further public health protection and that the LRAA alternative in combination with the IDSE is a balanced and reasonable approach. Although it will not remove all DBP peaks (individual samples with values greater than the MCL), this proposed regulation will ensure that DBP exposures across a system's distribution system are further reduced, are more equitable, and may reduce cancer and reproductive and developmental risk.

The Advisory Committee discussions primarily focused on the relative magnitude of exposure reduction versus the expected impact on the water industry and its customers. Initially, this analysis compared expected reductions in DBP levels and predictions of treatment technology changes associated with a wide variety of Stage 2 DBPR MCL alternatives.

After initial discussions, EPA and the Advisory Committee primarily focused on four types of alternative rule scenarios.

Preferred Alternative.—MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as LRAAs. Bromate MCL of 0.010 mg/L.

Alternative 1.—MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as LRAAs. Bromate MCL of 0.005 mg/L.

Alternative 2.—MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as individual sample maximums (*i.e.*, no single sample could exceed the MCL). Bromate MCL of 0.010 mg/L.

Alternative 3.—MCLs of 0.040 mg/L TTHM and 0.030 mg/L HAA5 as RAAs. Bromate MCL of 0.010 mg/L.

EPA and the Advisory Committee, with assistance from the Technical Workgroup, conducted an in-depth analysis of these regulatory alternatives. In the process of evaluating alternatives, EPA and the Advisory Committee reviewed vast quantities of data and many analyses that addressed health effects, DBP occurrence, predicted reductions in DBP levels, predicted technology changes, and capital, annual, and household costs. Details of the compliance, occurrence, and cost forecasts for the four alternative rule scenarios are described in the Stage 2 DBPR Economic Analysis (EA) (USEPA 2003i) and the Stage 2 DBPR Occurrence Document (USEPA 2003o).

In the end, the Advisory Committee recommended the Preferred Alternative in combination with the IDSE which they believed would reduce exposure to high levels of DBPs. Today, EPA is proposing the Preferred Alternative in combination with the IDSE.

The only difference between the Preferred Alternative and Alternative 1 is the bromate MCL. The Advisory Committee's recommendation to maintain the Stage 1 DBPR bromate MCL of 0.010 mg/L is discussed in section V.G. of today's proposal.

Alternatives 2 and 3 are significantly more stringent than the Stage 1 DBPR with respect to the TTHM and HAA5 requirements. Alternative 2 would require that all samples be below the MCL. Because DBP occurrence is variable across the distribution system and over time (as discussed in section IV), systems would have to base their disinfectant and treatment strategies on controlling their highest DBP occurrence levels. Alternative 3 maintains the Stage 1 DBPR RAA compliance calculation, but reduces the Stage 1 DBPR MCLs by 50 percent. Both alternatives 2 and 3 would cause significant changes in treatment for a large number of systems. The estimated costs for Alternatives 2 and 3 are approximately an order of magnitude above the costs for the Preferred Alternative (see section VII.B.).

Consistent with this greater stringency of alternatives 2 and 3, the predicted DBP reductions and the resulting health benefits for them are greater than those predicted for the Preferred Alternative. Although all members of the Advisory Committee believed that the science showing reproductive and developmental health effects that have been associated with DBPs was sufficient to cause concern and warrant regulatory action, the Advisory Committee did not believe that the association was certain enough to justify the substantial change in treatment technologies that would be required to meet these alternatives. Thus, the Advisory Committee rejected Alternatives 2 and 3.

c. *Basis for the LRAA.* This section discusses the data and information EPA used to determine that the LRAA is an appropriate compliance strategy for today's proposed rule. EPA has chosen compliance based on an LRAA due to concerns about levels of DBPs above the MCL in some portions of the distribution system. The LRAA standard will eliminate system-wide averaging. The individuals served in areas of the distribution system with above average DBP occurrence levels masked by averaging under an RAA are not receiving the same level of health protection. Although an LRAA standard still allows averaging at a single location over an annual period, EPA believes that changing the basis of compliance from an RAA to an LRAA will result in decreased exposure to above average DBP levels (*see* section VII.A. for predictions of DBP reductions under the LRAA MCLs). This conclusion is based on three considerations:

(1) There is considerable evidence that under the current RAA MCL compliance monitoring requirements a small but significant proportion of monitoring locations experience high DBP levels. As summarized in section IV of this preamble, 14 and 21% of Information Collection Rule systems currently meeting the Stage 1 DBPR RAA MCLs had TTHM and HAA5 single sample concentrations greater than the Stage 1 MCLs and ranged up to 140 μg/L and 130 μg/L respectively (Figures IV–1 and IV–2), though most of these exceedences were below 100 μg/L.

(2) In some situations, the populations served by certain portions of the distribution system consistently receive water that exceeds the MCL even though the system is in compliance. As discussed in section IV of this preamble, some Information Collection Rule systems meeting the Stage 1 DBPR RAA MCLs had monitoring locations that exceeded 0.080 mg/L TTHM and/or 0.060 mg/L HAA5 as an annual average (*i.e.*, as LRAAs) by up to 25% (Figures IV–3 and IV–4). Five percent of plants that achieved compliance with the Stage 1 TTHM MCL of 0.080 mg/L based on an RAA had a particular sampling location that exceeded 0.080 mg/L as an LRAA (Figure IV–3). Figure IV–4 shows similar results based on Information Collection Rule HAA5 data. Three percent of plants that met the Stage 1 HAA5 MCL of 0.060 mg/L as an RAA had a sampling location that exceeded 0.060 mg/L as an LRAA. Customers served at these locations consistently received water with TTHM and/or HAA5 concentrations higher than the system-wide MCL.

(3) Compliance based on an LRAA will remove the opportunity for systems to average out samples from high and low quality water sources. Some systems are able to comply with an RAA MCL even if they have a plant with a poor quality water source (that thus produces high concentrations of DBPs) because they have another plant that has a better quality water source (and thus lower concentrations of DBPs). Individuals served by the plant with the poor quality source will usually have higher DBP exposure than individuals served by the other plant.

d. *Basis for phasing LRAA compliance.* EPA believes that a phased approach for LRAA implementation will facilitate transition to the new compliance requirements. Stage 2A of this proposed rule does not require systems to conduct any additional monitoring. They will continue to monitor at Stage 1 DBPR locations. Because the LRAA calculation is the same as the RAA calculation if there is only one site, Stage 2A compliance only applies to systems that monitor at more than one site and will only affect medium and large surface water systems (serving at least 10,000 people) or systems with multiple plants. Thus, the majority of ground water systems, small surface water systems, and some consecutive systems are not affected by the proposed Stage 2A requirements.

e. *TTHM and HAA5 as Indicators.* In part, both the TTHM and HAA5 classes are regulated because they occur at high levels and represent chlorination byproducts that are produced from source waters with a wide range of water quality. The combination of TTHM and HAA5 represent a wide variety of compounds resulting from bromine substitution and chlorine substitution reactions (*i.e.*, bromoform has 3 bromines, TCAA has 3 chlorines, BDCM has one bromine and two chlorines, etc). EPA believes that the TTHM and HAA5 classes serve as an indicator for unidentified and unregulated DBPs. EPA believes that controlling the occurrence levels of TTHM and HAA5 will control the levels of all chlorination DBPs to some extent.

3. Request for Comment

EPA requests comment on the alternative MCL strategies that were considered by the Advisory Committee and the determination to propose the Preferred Alternative in combination with the IDSE as the preferred regulatory strategy. EPA also requests comment on whether the proposed approach will reduce peak DBP levels.

EPA requests comment on the phased MCL strategy and whether or not it will facilitate compliance with the LRAA. EPA also requests comment on the Stage 2A MCLs of 0.120 mg/L TTHM and 0.100 mg/L HAA5 as LRAAs and on the long-term MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as LRAAs.

E. Requirements for Peak TTHM and HAA5 Levels

1. What Is EPA Proposing Today?

Today, EPA is proposing that, concurrent with Stage 2B, systems must specifically document occurrences of peak DBP levels, termed significant excursions. In support of this provision, EPA is proposing that States, as a special primacy condition, develop criteria for determining whether a system has a significant excursion. EPA has developed draft guidance for systems and States on how systems may determine whether they have significant excursions. EPA is also proposing that a system that has a significant excursion must: (1) Evaluate distribution system operational practices to identify opportunities to reduce DBP levels (such as tank management to reduce residence time and flushing programs to reduce disinfectant demand), (2) prepare a written report of the evaluation, and (3) no later than the next sanitary survey, review the evaluation with their State. This review will take place under the sanitary survey components calling for the State to review monitoring, reporting, and data verification and system management and operation.

2. How Was This Proposal Developed?

Because individual measurements from a location are averaged over a four-quarter period to determine compliance, there may be occurrence levels that exceed the MCL even when a system is in compliance with an LRAA MCL. EPA and the Advisory Committee were concerned about these exposures to peak levels of DBPs and the possible risk they might pose. This concern was clearly reflected in the Agreement in Principle, which states,

''Recognizing that significant excursions of DBP levels will sometimes occur, even when systems are in full compliance with the enforceable MCL, public water systems that have significant excursions during peak periods are to refer to EPA guidance on how to conduct peak excursion evaluations, and how to reduce such peaks. Such excursions will be reviewed as part of the sanitary survey process. EPA guidance on DBP level excursions will be issued prior to promulgation of the final rule and will be developed in consultation with stakeholders.''

In evaluating this recommendation, EPA believes that the Advisory Committee's intent was clear with regard to the need for guidance on how to evaluate and reduce significant excursions. However, the Agreement is less clear on how, and where, to define what constitutes a significant excursion, and how to define the scope of the evaluation. EPA draft guidance recommends several approaches for determining whether significant excursions have occurred. While today's proposal requires an evaluation only of distribution system operational practices, EPA believes that many systems would benefit from a broader evaluation that includes treatment plant and other system operations.

EPA recognizes that different stakeholders have different points of view on whether specific criteria that initiate the evaluation of significant excursions should be included in the rule or in guidance. EPA also recognizes that different stakeholders may have different perspectives on how to identify a significant excursion. For this proposal, EPA has prepared draft guidance for systems and States on how to (1) determine whether a significant excursion has occurred, using several different options, (2) conduct significant excursion evaluations, and (3) reduce significant excursion occurrence.

3. Request for Comment

EPA requests comment on the proposed approach for addressing significant excursions and on the draft guidance. Is a special primacy condition the appropriate means for allowing flexibility in identifying significant excursions while ensuring that such evaluations occur? Is the sanitary survey the appropriate mechanism for reviewing significant excursion data with the State? Should a system be required to take corrective action when significant excursions occur? Should the required scope of the evaluation be expanded beyond distribution system operations?

EPA also requests comment on whether specific criteria that initiate the evaluation of significant excursions should be included in the rule or in guidance. EPA requests comment on how to identify significant excursions (regardless of whether the criteria are in the rule or in guidance). For example, should the significant excursion be based on an individual measurement, *e.g.*, any measurement being 25 or 50% over either the TTHM or HAA5 MCLs? Alternatively, should the determination of a significant excursion be based on a certain level of variability among multiple measurements? For example,

should the significant excursion be based on the standard deviation of the LRAA exceeding specific numerical values for either TTHM (*e.g.*, 0.020 mg/l) or HAA5 (*e.g.*, 0.015 mg/L)? Or should the excursion be based on a relative measure of variability (*e.g.*, a relative standard deviation exceeding 25% or 50%) with the condition of a threshold average concentration also being exceeded (*e.g.*, an LRAA needing to be at least 0.040 mg/l for TTHM or 0.030 mg/l for HAA5)? EPA requests comment on the above approaches or alternative approaches for determining whether a significant excursion has occurred. EPA also requests comment on whether different approaches may be appropriate for large and small systems.

F. BAT for TTHM and HAA5

1. What Is EPA Proposing Today?

Today, EPA is proposing that the best available technology (BAT) for the TTHM and HAA5 LRAA MCLs (0.080 mg/L and 0.060 mg/L respectively) be one of the three following technologies:

(1) GAC adsorbers with at least 10 minutes of empty bed contact time and an annual average reactivation/replacement frequency no greater than 120 days, plus enhanced coagulation or enhanced softening.

(2) GAC adsorbers with at least 20 minutes of empty bed contact time and an annual average reactivation/replacement frequency no greater than 240 days.

(3) Nanofiltration (NF) using a membrane with a molecular weight cut off of 1000 Daltons or less (or demonstrated to reject at least 80% of the influent TOC concentration under typical operating conditions).

EPA is proposing a different BAT for consecutive systems than for wholesale systems to meet the TTHM and HAA5 LRAA MCLs. The proposed consecutive system BAT is chloramination with management of hydraulic flow and storage to minimize residence time in the distribution system.

2. How Was This Proposal Developed?

a. *Basis for the BAT.* The Safe Drinking Water Act directs EPA to specify BAT for use in achieving compliance with the MCL. Systems unable to meet the MCL after application of BAT can get a variance (*see* section V.L. for a discussion of variances). Systems are not required to use BAT in order to comply with the MCL. They can use other technologies as long as they meet all drinking water standards and are approved by the State.

EPA examined BAT using two different methods: (1) EPA analyzed data from the Information Collection Rule treatment studies and (2) EPA used the Surface Water Analytical Tool (SWAT), a model developed to compare alternative regulatory strategies. Both analyses support the BAT options proposed today. The results of each analyses are presented in the following two sections.

i. BAT analysis using the Information Collection Rule treatment studies. EPA analyzed data from the Information Collection Rule treatment studies (Information Collection Rule Treatment Study Database CD–ROM, Version 1.0, USEPA 2000m; Hooper and Allgeier 2002). The treatment studies were designed to evaluate the technical feasibility of using GAC and NF to remove DBP precursors prior to the addition of chlorine-based disinfectants. Systems were required to conduct an Information Collection Rule treatment study based on TOC levels in the source or finished water. Specifically, surface water plants with annual average source water TOC concentrations greater than 4 mg/L and ground water plants with annual average finished water TOC concentrations greater than 2 mg/L were required to conduct treatment studies. Thus, the plants required to conduct treatment studies generally had waters with organic DBP precursor levels that were significantly higher than the Information Collection Rule national plant medians of 2.7 mg/L for source water at surface water plants and 0.2 mg/L for finished water at ground water plants (USEPA 2003o).

Plants that conducted GAC studies typically evaluated performance at two empty bed contact times, 10 and 20 minutes, over a wide range of operational run times to evaluate the variable nature of TOC removal by GAC. This allowed GAC performance to be assessed with respect to empty bed contact time as well as reactivation/replacement frequency. Plants that conducted membrane treatment studies evaluated one or two nanofiltration membranes with molecular weight cutoffs less than 1000 Daltons. Regardless of the technology evaluated, all treatment studies evaluated DBP formation in the effluent from the advanced process under simulated distribution system conditions representative of the average residence time and using free chlorine as the primary and residual disinfectant. (For more information on the Information Collection Rule treatment study requirements and testing protocols, see USEPA 1996 a and b.)

Based on the treatment study results, GAC is effective for controlling DBP formation for waters with influent TOC concentrations below approximately 6 mg/L (based on the Information Collection Rule and NRWA data, over 90 percent of plants have average influent TOC levels below 6 mg/L (USEPA 2003o)). Of the plants that conducted an Information Collection Rule GAC treatment study, approximately 70% of the surface water plants studies could meet the 0.080 mg/L TTHM and 0.060 mg/L HAA5 MCLs, with a 20% safety factor (*i.e.*, 0.064 mg/L and 0.048 mg/L, respectively) using GAC with 10 minutes of empty bed contact time and a 120 day reactivation frequency, and 78% of the plants could meet the MCLs with a 20% safety factor using GAC with 20 minutes of empty bed contact time and a 240 day reactivation frequency. As discussed previously, the treatment studies were conducted at plants with poorer water quality than the national average. Therefore, EPA believes that much higher percentages of plants nationwide could meet the MCLs with the proposed GAC BATs.

Among plants using GAC, larger systems would likely realize an economic benefit from on-site reactivation, which could allow them to use smaller, 10-minute empty bed contact time contactors with more frequent reactivation (*i.e.*, 120 days or less). Most small systems would not find it economically advantageous to install on-site carbon reactivation facilities, and thus would opt for larger, 20-minute empty bed contact time contactors, with less frequent carbon replacement (*i.e.*, 240 days or less).

The proposed reactivation/replacement interval for the 20 minute contactor (*i.e.*, 240 days) is double the reactivation/replacement interval for 10 minute contactor (*i.e.*, 120 days). This is based on the assumption of a linear relationship between empty bed contact time and the reactivation interval (*e.g.*, a doubling of the empty bed contact time will result in a doubling of the reactivation interval). The data from the Information Collection Rule treatment studies indicates that this linear relationship may not always hold and that doubling the empty bed contact time generally results in more than a doubling of the reactivation interval. While there may be some operational advantage in using larger empty bed contact times, the larger contactors will result in additional capital expenditures. Furthermore, the economic optimization of a GAC process must also consider the number of smaller contactors in parallel, since it may be advantageous to operate a larger number of smaller contactors in parallel, allowing each individual contactor to be

operated for a longer period of time. Based on these considerations, and the analysis of subject matter experts, it was concluded that the proposed combination of GAC empty bed contact times and reactivation/replacement intervals were reasonable for BAT.

The Information Collection Rule treatment study results also demonstrated that nanofiltration was the better DBP control technology for ground water sources with high TOC concentrations (*i.e.*, above approximately 6 mg/L). The results of the membrane treatment studies showed that all ground water plants could meet the 0.080 mg/L TTHM and 0.060 mg/L HAA5 MCLs, with a 20% safety factor (*i.e.*, 0.064 mg/L and 0.048 mg/L, respectively) at the average distribution system residence time using nanofiltration. Nanofiltration would be less expensive than GAC for high TOC ground waters, which generally require minimal pretreatment prior to the membrane process. Also, nanofiltration is an accepted technology for treatment of high TOC ground waters in Florida and parts of the Southwest, areas of the country with elevated TOC levels in ground waters.

ii. *BAT analysis using the SWAT.* The second method that EPA used to examine alternatives for BAT was the SWAT model that was developed to compare alternative regulatory strategies. EPA modeled the following BAT options: enhanced coagulation/softening with chlorine (the Stage 1 DBPR BAT); enhanced coagulation/softening with chlorine and no predisinfection; enhanced coagulation and GAC10; enhanced coagulation and GAC20; and enhanced coagulation and chloramines. Enhanced coagulation/softening is required under the Stage 1 DBPR at subpart H conventional filtration plants. In the model, GAC10 was defined as granular activated carbon with an empty bed contact time of 10 minutes and a reactivation or replacement interval of 90 days or longer. GAC20 was defined as granular activated carbon with an empty bed contact time of 20 minutes and a reactivation or replacement interval of 90 days or longer. EPA assumed that systems would be operating to achieve both the Stage 2B MCLs of 0.080 mg/L TTHM and 0.060 mg/L HAA5 as an LRAA and the SWTR removal and inactivation requirements of 3-log for *Giardia* and 4-log for viruses. EPA also evaluated the BAT options under the assumption that plants operate to achieve DBP levels 20% below the MCL (safety factor). These assumptions along with other inputs for the SWAT runs are consistent with those used in the Economic Analysis of today's proposed rule (USEPA 2003i).

The compliance percentages forecasted by the SWAT model are indicated in Table V–1. EPA estimates that more than 97% of large systems will be able to achieve the Stage 2B MCLs regardless of post-disinfection choice if they were to apply one of the proposed GAC BATs, *i.e.*, enhanced coagulation (EC) and GAC10 (Seidel Memo, 2001). As shown in the Stage 2 DBPR Occurrence document (USEPA 2003o), the source water quality (*e.g.*, DBP precursor levels) in medium and small systems is expected to be comparable to or better than that for the large systems. Based on the large system estimate, EPA believes it is conservative to assume that at least 90% of medium and small systems will be able to achieve the Stage 2B MCLs if they were to apply one of the proposed GAC BATs. EPA assumes that small systems may adopt GAC20 in a replacement mode (with replacement every 240 days) over GAC10 because it may not be economically feasible for some small systems to install and operate an on-site GAC reactivation facility. Moreover, some small systems may find nanofiltration cheaper than the GAC20 in a replacement mode if their specific geographic locations cause a relatively high cost for routine GAC shipment.

TABLE V–1.—SWAT MODEL PREDICTIONS OF PERCENT OF LARGE PLANTS IN COMPLIANCE WITH TTHM AND HAA5 STAGE 2B MCLS AFTER APPLICATION OF SPECIFIED TREATMENT TECHNOLOGIES

Technology*	Compliance with 0.080 mg/L (TTHM)/0.060 mg/L (HAA5) LRAAs			Compliance with 0.064 mg/L (TTHM)/0.048 mg/L (HAA5) LRAAs (MCLs with 20% safety factor)		
	Residual disinfectant		All systems	Residual disinfectant		All systems
	Chlorine	Chloramine		Chlorine	Chloramine	
Enhanced Coagulation (EC)	73.5	76.9	74.8	57.2	65.4	60.4
EC (no predisinfection)	73.4	88.0	78.4	44.1	62.7	50.5
EC & GAC10	100	97.1	99.1	100	95.7	98.6
EC & GAC20	100	100	100	100	100	100
EC & All Chloramines	NA	83.9	NA	NA	73.6	NA

* Enhanced coagulation/softening is required under the Stage 1 DBPR for conventional plants.

b. *Basis for the Consecutive System BAT.* EPA believes that the best compliance strategy for consecutive systems is to collaborate with wholesalers on the water quality they need. For consecutive systems that are having difficulty meeting the MCLs, EPA is proposing a BAT of chloramination with management of hydraulic flow and storage to minimize residence time in the distribution system. EPA is proposing a different BAT than for wholesale systems because a consecutive system's source water has already been disinfected and contains DBPs that cannot be effectively removed or controlled with the BATs proposed for wholesale systems. EPA believes the proposed consecutive system BAT is an effective means for consecutive systems to meet the MCLs.

Chloramination has been used for residual disinfection for many years to minimize the formation of chlorination DBPs, including TTHM and HAA5 (Stage 2 Technology and Cost Document, USEPA 2003k). The BAT provision to manage hydraulic flow and minimize residence time in the distribution system is to facilitate the maintenance of the chloramine residual and minimize the likelihood for nitrification. Nitrification, the process by which microbes convert free ammonia to nitrate and nitrite, is a concern for systems using chloramines. Nitrification, however, can be controlled with appropriate chlorine to ammonia ratios, increasing flow in low demand areas, and increasing storage tank turnover. EPA proposes that systems implementing the consecutive system BAT must do the following: (1) Maintain a chloramine residual throughout the distribution system, (2) develop and submit a plan that indicates actions that will be taken to minimize the residence time of water

within the distribution system, (3) have the plan approved by the Primacy Agency, and (4) implement the plan as approved by the Primacy Agency. Minimum components of the management plan would include periodic scheduled flushing of all dead end pipes and storage vessels through which water is delivered to customers, and hydraulic flow control procedures that routinely circulate water in all storage vessels within the distribution system.

EPA believes that the BATs proposed for wholesale systems are not appropriate for consecutive systems because each of these BATs, when applied to water with DBPs, raises other concerns. GAC is not cost-effective for removing DBPs. In addition, dioxin, a carcinogen, may be formed during GAC regeneration if GAC has been used to adsorb chlorinated DBPs. Nanofiltration is only moderately effective at removing THMs or HAAs if membranes that have a very low molecular weight cutoff and very high cost of operation are employed. Therefore, GAC and nanofiltration are not appropriate BATs for consecutive systems.

3. Request for Comment

EPA requests comment on the proposed BATs including the BAT for consecutive systems.

G. MCL, BAT, and Monitoring for Bromate

1. What Is EPA Proposing Today?

EPA is proposing today that the MCL for bromate for systems using ozone remain at 0.010 mg/L as an RAA for samples taken at the entrance to the distribution system as established by the Stage 1 DBPR and as provided for under the risk-balancing provisions of section 1412(b)(5) of the SDWA. EPA's proposal is consistent with the recommendation of the Stage 2 M–DBP Advisory Committee, which considered the potential that reducing the bromate MCL could both increase the concentration of other DBPs in the drinking water and interfere with the efficacy of microbial pathogen inactivation. In addition, as required by the SDWA and as recommended by the Advisory Committee, EPA will review the bromate MCL as part of the 6-year review process and determine whether the MCL should remain at 0.010 mg/L or be reduced to a lower level. As a part of that review, EPA will consider the increased utilization of alternative technologies, such as UV, and whether the risk/risk concerns reflected in today's proposal remain valid.

Because EPA is not revising the Stage 1 DBPR bromate MCL, EPA is not proposing a revised BAT for bromate. The Stage 1 DBPR BAT for bromate is defined as control of ozone treatment processes to reduce production of bromate. EPA also determined that it was not necessary to regulate bromate in non-ozone systems that use hypochlorite.

Finally, EPA is proposing to modify the criterion for a system that uses ozone (and therefore must monitor for bromate) to qualify for reduced bromate monitoring from one sample per ozone plant per month to one sample per plant per quarter.

2. How Was This Proposal Developed?

a. *Bromate MCL.* Bromate is a principal byproduct from ozonation of bromide-containing source waters. As described in more detail later, making the bromate MCL more stringent has the potential to decrease current levels of microbial protection, impair the ability of systems to control resistant pathogens like *Cryptosporidium,* and increase levels of DBPs from other disinfectants that may be used instead of ozone.

EPA estimates that the 1 in 10,000 excess lifetime cancer risk level for bromate is 0.005 mg/L. EPA proposed and ultimately finalized an MCL of 0.010 mg/L in the Stage 1 DBPR, primarily because available analytical detection methods for bromate could only reliably measure to 0.01 mg/L (USEPA 1994b). Analytical methods for bromate are now available to quantify bromate concentrations as low as 0.001 mg/L. Due to the availability of lower detection methods for bromate, as part of the Stage 2 M–DBP Advisory Committee deliberations, EPA considered revising the MCL to 0.005 mg/L or lower.

As a disinfectant, ozone is highly effective against a broad range of microbial pathogens including bacteria, viruses, and protozoa. Moreover, ozone is one of the few disinfectants available in water treatment that is capable of inactivating *Cryptosporidium,* a protozoan which can cause severe intestinal disorders and can be deadly to those with compromised immune systems. The oxidizing properties of ozone are also valuable for treatment objectives like control of tastes and odors and removal of iron and manganese. In contrast, chlorine, the most common disinfectant and oxidant in water treatment, is substantially less effective for controlling *Cryptosporidium.* Chlorine dioxide, while capable of providing low levels of inactivation for *Cryptosporidium,* typically cannot be used at high doses without violating the MCL for chlorite, a byproduct of chlorine dioxide. UV light is highly effective against *Cryptosporidium* and *Giardia* and most viruses, but has not been used extensively to treat drinking water in the United States.

As of early 2000, there were 332 plants of various sizes using ozone (Overbeck 2000) and 58 plants that were planning to install ozonation (Rice 2000—personal communication: email 7/14/2000). A significant percent of current ozone plants use ozone for some portion of their disinfection objective (Rice, 2000—personal communication: email 7/14/2000). An ozone system that could not meet a 0.005 mg/L bromate MCL would have three primary options: decrease the ozone dose; switch to a different disinfectant; or install an advanced filtration process such as membranes, sometimes in combination with the first two options. Of these three options, the third is likely effective but very expensive, while the first two create the risk either of reducing microbial protection for a wide range of microbial pathogens, or of increasing formation of DBPs other than bromate.

In an attempt to achieve a lower level of bromate, some systems might be driven to reduce the applied ozone dose to the minimum necessary for regulatory compliance or switch to other treatment processes. Many systems currently achieve more disinfection than is required by the SWTR and if a system were to simply lower the ozone dose, protection from pathogens may be compromised. In addition, since inactivation of *Cryptosporidium* requires much higher ozone doses than *Giardia* inactivation, systems cannot achieve *Cryptosporidium* inactivation with low ozone doses.

If a system were to lower the ozone dose and supplement with an additional disinfectant, or switch entirely to a different disinfectant, the system may not achieve the same level of microbial protection as is afforded by ozonation. Also, other potentially harmful byproducts from the different disinfectant would be produced.

During the Stage 2 M–DBP Advisory Committee discussions, the TWG evaluated the impact of reducing the bromate MCL from 0.010 mg/L to 0.005 mg/L as an annual average. The TWG concluded that many systems currently using ozone or predicted to install ozone to inactivate microbial pathogens would have significant difficulty maintaining bromate levels at or below 0.005 mg/L. In the Information Collection Rule survey of systems serving greater than 100,000 people, all of the ozone plants had annual average

bromate concentrations below the 0.010 mg/L level (USEPA 2003o). However, approximately 20% of these ozone plants did not meet the 0.005 mg/L level. Using the assumption that systems operate their plants using a safety margin of 20% below the MCL, about 30% of ozone plants did not reliably attain this level (0.004 mg/L). During the Information Collection Rule, for the first half of 1998, much of the U.S. was wetter than normal (NOAA 1998). This hydrogeological condition often leads to lower than normal bromide concentrations due to dilution by higher water flows. In the second half of 1998, California continued to experience El Nino rains (40% of Information Collection Rule ozone plants were located in California) but many other areas of the country such as Texas and Florida experienced a drought. The percentage of ozone systems unable to achieve 0.005 mg/L bromate would likely increase during years in which bromide concentrations in California were elevated as consequence of drought.

The ability of systems to use ozone to meet *Cryptosporidium* treatment requirements proposed under the LT2ESWTR would be diminished if the bromate MCL was decreased from 0.010 to 0.005 mg/L. The proposed LT2ESWTR will require a subset of systems, based on source water pathogen levels, to provide from 1.0 to 2.5 logs of additional treatment for *Cryptosporidium*. Ozone doses required to inactivate *Cryptosporidium* are substantially greater than those required for Giardia and viruses. To assess the potential impact of a lower bromate MCL on the ability of systems to treat for *Cryptosporidium*, the TWG estimated the percentage of treatment plants that could use ozone to inactivate from 0.5 to 2.5 log of *Cryptosporidium* without exceeding a bromate MCL of either 0.005 or 0.010 mg/L (USEPA 2003i). These estimations were based on analyses of Information Collection Rule source water quality data, coupled with projected ozone dose requirements for *Cryptosporidium*. This analysis suggests that 88% of systems could use ozone to achieve 1 log of *Cryptosporidium* inactivation and 47% could inactivate 2 log while complying with a bromate MCL of 0.010 mg/L. With the bromate MCL reduced to 0.005 mg/L, though, these estimates drop to 67% of systems able to inactivate 1 log of *Cryptosporidium* with ozone and only 14% able to inactivate 2 log. The number of plants predicted to be able to treat for *Cryptosporidium* with ozone and meet a 0.005 mg/L standard was further reduced when periods of higher bromide levels, similar to drought conditions, were modeled. This trend is further exacerbated since the proposed LT2ESWTR would require more stringent ozone operating conditions (such as higher ozone doses and longer contact times) than under current surface water treatment requirements for the subset of plants with higher *Cryptosporidium* concentrations in their source water and would thus result in higher bromate formation than assumed by the TWG. Thus, as systems are required to meet more stringent inactivation requirements, a large number of systems would be forced to select treatment processes other than ozone if the bromate standard were lowered to 0.005 mg/L.

The Stage 2 M–DBP Advisory Committee considered that reducing the bromate MCL to 0.005 mg/L could both increase the concentration of other DBPs in the drinking water and interfere with the efficacy of microbial pathogen inactivation. Therefore, the Advisory Committee recommended, for purposes of the Stage 2 DBPR, that the bromate MCL remain at 0.010 mg/L. EPA will review the bromate MCL as part of the ongoing 6-year review process and determine whether the MCL should remain at 0.010 mg/L or be reduced to a lower concentration based on new information.

Today, EPA is proposing to leave the bromate MCL at 0.010 mg/L, consistent with the Advisory Committee's recommendation. EPA believes that this is a prudent step at this time, in order to preserve microbial protection. EPA will continue to analyze any new bromate health effects data as they become available. It is possible that EPA may determine that the bromate MCL should be decreased to 0.005 mg/L or lower in a future rulemaking.

b. *Bromate in hypochlorite solutions.* The Stage 2 M–DBP Advisory Committee also discussed the issue of hypochlorite solutions contaminated with bromate. This contamination can occur during the production of hypochlorite solutions from natural salt deposits. The range of bromate concentrations in hypochlorite stock solutions varies widely (Bolyard *et al.* 1992; Chlorine Institute 1999, 2000). Moreover, the bromate contained in the stock solution is diluted upon addition to the drinking water. From data on Information Collection Rule ozone systems that used hypochlorite versus those that used gaseous chlorine, the TWG estimated that hypochlorite solutions contributed an average of 0.001 mg/L bromate.

The Advisory Committee discussed these results and, since the bromate level resulting from hypochlorite solutions was small compared to the MCL, did not recommend regulating bromate at systems not using ozone (non-ozone systems). The Advisory Committee recognized that ozone systems also using hypochlorite will have to be careful about the quality of their stock solution.

c. *Criterion for reduced bromate monitoring.* Because more sensitive bromate methods are now available, EPA is proposing a new criterion for reduced bromate monitoring. In the Stage 1 DBPR, EPA required ozone systems to demonstrate that source water bromide levels, as a running annual average, did not exceed 0.05 mg/L. EPA elected to use bromide as a surrogate for bromate in determining eligibility for reduced monitoring because the available analytical method for bromate was not sensitive enough to quantify levels well below the bromate MCL of 0.010 mg/L.

In section V.O., EPA is proposing several new analytical methods for bromate that are far more sensitive than the existing method. Since these methods can measure bromate to levels of 0.001 mg/L or lower, EPA is proposing to replace the criterion for reduced bromate monitoring (source water bromide running annual average not to exceed 0.05 mg/L) with a bromate running annual average not to exceed 0.0025 mg/L.

In the past, EPA has often set the criterion for reduced monitoring eligibility at 50% of the MCL, which would be 0.005 mg/L. However, as discussed before, EPA is proposing that the MCL for bromate remain at 0.010 mg/L, a level that is higher than EPA's usual excess cancer risk range of 10(-4) to 10(-6) at 2x10(-4) because of risk tradeoff considerations. EPA believes that the decision for reduced monitoring is separate from these risk tradeoff considerations. Risk tradeoff considerations influence the selection of the MCL, while reduced monitoring requirements are designed to ensure that the MCL, once established, is reliably and consistently achieved. Requiring a running annual average of 0.0025 mg/L for the reduced monitoring criterion allows greater confidence that the system is achieving the MCL and thus ensuring public health protection.

3. Request for Comment

EPA requests comment on the decision to maintain the Stage 1 DBPR bromate BAT and MCL of 0.010 mg/L. EPA also requests comment on the decision not to require bromate

monitoring at non-ozone systems that use hypochlorite.

EPA requests comment on whether the criterion for reduced bromate monitoring should be set at a level other than 0.0025 mg/L, and a rationale for setting it at that level.

H. Initial Distribution System Evaluation (IDSE)

The IDSE is an important part of today's proposed regulation that is intended to identify sample locations for Stage 2B compliance monitoring that represent distribution system sites with high DBP concentrations.

1. What is EPA Proposing Today?

EPA is proposing a requirement for systems to perform an Initial Distribution System Evaluation (IDSE). Systems will collect data on DBP levels throughout their distribution system, evaluate these data to determine which sampling locations are most representative of high DBP levels and compile this information into a report for submission to the primacy agency.

a. *Applicability.* All community water systems, and large nontransient noncommunity water systems (those serving at least 10,000 people) that add a primary or residual disinfectant other than ultraviolet light, or that deliver water that has been treated with a primary or residual disinfectant other than ultraviolet light (*i.e.*, consecutive systems) are required to conduct an IDSE under the proposed rule. The IDSE requirement for systems serving fewer than 500 people may be waived if the State determines that the monitoring site approved for Stage 1 DBPR compliance is sufficient to represent both high HAA5 and high TTHM concentrations. The State must submit criteria for this waiver determination to EPA as part of their primacy application. States may decide to waive the IDSE requirement for all systems serving fewer than 500 or some subset of all systems serving fewer than 500 if the State determines that it is appropriate. EPA is developing an IDSE Guidance Manual that will include guidance to States on situations for which a waiver would be appropriate (USEPA 2003j).

b. *Data collection.* IDSEs are intended to help identify and select Stage 2B compliance monitoring sites that represent high concentrations of TTHMs and HAA5. To be able to identify these sites, systems and States must have monitoring data collected from throughout their distribution systems. Therefore, under today's proposed rule, systems are required to collect monitoring data on the concentrations of these DBPs. There are three possible approaches by which a system can meet the IDSE requirement.

i. *Standard monitoring program.* The standard monitoring program requires one year of monitoring on a specified schedule throughout the distribution system. The frequency and number of samples required under the standard monitoring program is determined by source water type, number of treatment plants, and system size (see section V.J. for a more detailed discussion of the specific monitoring requirements). Prior to commencing the standard monitoring program, systems must prepare a monitoring plan. EPA's IDSE Guidance Manual will provide guidance on selecting monitoring sites and conducting the standard monitoring program (USEPA 2003j). As recommended by the Advisory Committee, EPA is proposing that the standard monitoring program results are not to be used for determining compliance with MCLs and that systems will not be required to report IDSE results in the Consumer Confidence Report.

ii. *System specific study.* Under this approach, systems may choose to perform a system-specific study based on earlier monitoring studies or other data analysis in lieu of the standard monitoring program. These studies must provide equivalent or better information than the standard monitoring program for selecting sites that represent high TTHM and HAA5 levels. Examples of alternative studies are: (1) Recent TTHM and HAA5 monitoring data that encompass a wide range of sample sites representative of the distribution system, including those judged to represent high TTHM and HAA5 concentrations and (2) hydraulic modeling studies that simulate water movement in the distribution system. Historical TTHM and HAA5 results submitted by systems must have been generated by certified laboratories and must include the system's most recent data. Treatment plant and distribution system characteristics at the time of historical data collection must reflect the current plant operations and distribution system. EPA's IDSE Guidance Manual will include a guidance for system-specific studies and how to determine whether site-specific data could be sufficient to meet the IDSE requirements (USEPA 2003j).

iii. *40/30 certification.* Under this approach, systems certify to their primacy agency that all required Stage 1 DBPR compliance samples were properly collected and analyzed during the two years prior to the start of the IDSE, and all individual compliance samples were ≤ 0.040 mg/L for TTHM and ≤0.030 mg/L for HAA5. Properly collected and analyzed compliance samples are those taken at required locations at times specified in the system's Stage 1 DBPR monitoring plan and analyzed by certified laboratories. Systems not required to collect Stage 1 DBPR compliance samples can not utilize the 40/30 certification approach because they do not have data to determine sampling locations that represent high concentrations of TTHMs and HAA5. Systems that qualify for reduced monitoring for the Stage 1 DBPR during the two years prior to the start of the IDSE, may use results of both routine and reduced Stage 1 DBPR monitoring to prepare the 40/30 certification. Large ground water systems may not have two years of HAA5 data to evaluate due to the timing of the Stage 1 DBPR and the IDSE requirements. EPA is proposing that, if two years worth of HAA5 data are not available, large ground water systems evaluate the most recent two years of TTHM data including data collected in accordance with the 1979 TTHM rule and all available HAA5 compliance data collected up to nine months following promulgation of this rule when making the 40/30 certification. Similarly, small wholesale and consecutive systems required to submit their IDSE report no later than two years after publication of the final rule will evaluate all available Stage 1 DBPR compliance data collected up to nine months following promulgation.

c. *Implementation.* All systems subject to the IDSE requirement under the proposed rule (except those receiving a very small system waiver from the State) must submit a report to the primacy agency. The requirements for the report depend upon the IDSE data collection alternative that the system selects and are listed in Table V–2.

TABLE V–2.—IDSE REPORT REQUIREMENTS

IDSE data collection alternative	IDSE report requirements
Standard Monitoring Program.	• All standard monitoring program TTHM and HAA5 analytical results, the original monitoring plan, and an explanation of any deviations from that plan. • A schematic of the distribution system. • Recommendations and justification for where and during what month(s) Stage 2B monitoring should be conducted.
System Specific Study	• All studies, reports, analytical results and modeling. • A schematic of the distribution system. • Recommendations and justification for where and during what month(s) Stage 2B monitoring should be conducted
40/30 Certification	• A certification that all required compliance samples were properly collected and analyzed during the two years prior to the start of the IDSE and all individual compliance samples were ≤ 0.040 mg/L for TTHM and ≤0.030 mg/L for HAA5. • Results of compliance samples taken after the IDSE was scheduled to begin and before the IDSE report was submitted. • Recommendations for where and during what month(s) Stage 2B monitoring should be conducted.

All IDSE reports must include recommendations for the location and schedule for the Stage 2B monitoring. The number of sampling locations and the criteria for their selection are described in § 141.605 of today's proposed rule, and in section V.I. Generally, a system must recommend locations with the highest LRAAs unless it provides a rationale (such as ensuring geographical coverage of the distribution system instead of clustering all sites in a particular section of the distribution system) for selecting other locations. Systems must consider both their compliance data and IDSE data in making this determination. In addition to specifying a protocol for identifying recommended monitoring sites in the rule language, EPA will provide guidance for recommending compliance monitoring sites (including rationales for systems to recommend sites that do not have the highest LRAA concentrations) and preparing the IDSE report. EPA will also provide a process to address IDSE implementation issues during the period prior to State primacy. At the time that systems serving fewer than 10,000 people conduct their monitoring or analyze their site-specific data, many States may have primacy.

The compliance schedules for the IDSE and other requirements of the proposed rule are described in detail in section V.J. Systems serving at least 10,000 people (and those smaller wholesale and consecutive systems associated with larger systems) will be collecting data for their IDSE prior to State primacy. EPA intends to have an IDSE Guidance Manual available to assist systems in performing the IDSE (USEPA 2003j). Primacy agencies will specify requirements for systems that do not submit an IDSE report, or that have not, in the determination of the primacy agency, conducted an adequate IDSE, in addition to giving the system a monitoring and reporting violation. These requirements may include repeating the IDSE while conducting compliance monitoring at Stage 1 monitoring sites or conducting Stage 2 compliance monitoring at sites selected by the State.

Consecutive systems are subject to the IDSE requirements of today's proposed rule. IDSE requirements for consecutive systems are largely the same as for other systems, but with two differences. First, the schedule for completion of the IDSE by a consecutive system is dependent upon the population of the wholesale system. If a consecutive system serving fewer than 10,000 buys water from a system that serves 10,000 or more people, then this consecutive system must comply within the same schedule as that for systems ≥ 10,000. Conversely, if a wholesale system serves < 10,000 but sells water to a consecutive system serving ≥ 10,000, then both the wholesale system and the consecutive system must complete the IDSE within the same schedule as that for systems ≥ 10,000. The second difference for consecutive systems is that the procedure for recommending Stage 2B compliance monitoring locations is modified for consecutive systems purchasing or receiving all of their finished water from a wholesale system. These modified procedures are described in § 141.605 of today's proposed rule, and in section V.I.

2. How Was This Pr oposal Developed?

The IDSE was recommended by the Stage 2 M–DBP Advisory Committee. The Advisory Committee believed that maintaining Stage 1 DBPR sampling sites for the Stage 2 DBPR would not accomplish the objective of providing consistent and equitable protection across the distribution system.

a. *Applicability.* The M–DBP Advisory Committee recommended that an IDSE be performed on all community systems to help to identify the locations in the distribution system that represent high DBP concentrations. EPA believes that large nontransient noncommunity water systems (those serving at least 10,000 people) also have distribution systems that require further evaluation to determine the most representative locations of high DBP levels. Therefore, large nontransient noncommunity systems and all community systems are required to perform an IDSE under today's proposal.

States may waive the IDSE requirement for those very small systems (systems that serve fewer than 500 people) that monitor for Stage 1 DBPR compliance at the maximum residence time site if the State determines their maximum residence time Stage 1 compliance monitoring site is likely to capture both the high TTHM and high HAA5 levels within the distribution system. The Advisory Committee recommended this waiver be included because many very small systems have small distribution systems and the high TTHM and high HAA5 site is at the same location. The Advisory Committee also recognized that not all very small systems have a single monitoring site that would represent both high TTHM and high HAA5 levels (*e.g.*, some rural systems with large distribution systems) and thus did not recommend a blanket IDSE waiver for all very small systems.

b. *Data collection.* The data collection requirements of the IDSE are designed to find both high TTHM and high HAA5 sites (*see* section V.I. for IDSE monitoring site locations). The IDSE is intended as a one-time requirement. High TTHM and HAA5 concentrations often occur at different locations in the

distribution system. The Stage 1 DBPR monitoring sites identified as the maximum location are selected according to residence time. Because HAAs can degrade in the distribution system in the absence of sufficient disinfectant residual (Baribeau *et al.* 2000), residence time alone is not an ideal criterion for identifying high HAA5 sites. The Information Collection Rule data show that of the four monitoring locations sampled per system, the one identified as the maximum residence time location was often not the location where the highest DBP levels were found. In fact, over 60 percent of the highest HAA5 LRAAs and 50 percent of the highest TTHM LRAAs were found at sampling locations in the system other than the maximum residence time location (*see* section IV). Thus the method and assumptions used to select the Information Collection Rule monitoring sites, and the Stage 1 DBPR compliance monitoring sites, are not sufficiently reliable to select Stage 2 DBPR compliance monitoring sites that will capture high DBP levels.

This data analysis reveals that a reevaluation of monitoring sites is necessary at many systems to capture sites with high DBP levels. The Advisory Committee recommended sample locations (based on distribution disinfectant type) at widely distributed sites (*see* section V.I. for details on IDSE monitoring requirements). Monitoring at additional sites across the distribution system increases the chance of finding sites with high DBP levels and targets both DBPs that degrade, and DBPs that form, as residence time increases in the distribution system. EPA believes that the required number of monitoring locations plus Stage 1 monitoring results provides an adequate recharacterization of DBP levels throughout the distribution system, at a reasonable cost. With a recharacterization of distribution systems that focuses on both high TTHM and HAA5 occurrence, EPA believes that high occurrence sites will be better represented in this standard monitoring program. Systems will be required to take steps to address high DBP levels at points that might otherwise have gone undetected. EPA believes that the decrease in DBP exposure anticipated to result from the transition from an RAA to an LRAA will be augmented by the IDSE.

The frequency and number of samples required for the standard monitoring program decrease as system size (population served) decreases and depend on source water type. The Advisory Committee believed that the number of samples required for large and medium surface water systems was not necessary for small surface water systems and ground water systems. The majority of small systems have distribution systems with simpler designs than large systems. DBP occurrence in ground water systems is generally lower and less variable than in surface water systems due to lower and less variable precursor levels and much less temperature variation (*see* section IV).

Committee members recognized that some systems have detailed knowledge of their distribution systems by way of hydraulic modeling and/or ongoing widespread monitoring plans (well beyond that required for compliance monitoring) that would provide equivalent or superior monitoring site selection compared to IDSE monitoring. Therefore, the Advisory Committee recommended that such systems be allowed to determine new monitoring sites using system-specific data such as historical monitoring data.

Systems that certify to their State that all compliance samples taken in the two years prior to the start of the IDSE were ≤ 0.040 mg/L TTHM and ≤ 0.030 mg/L HAA5 are not required to collect additional DBP monitoring data because the Advisory Committee determined that these systems most likely would not have high peak DBP levels. EPA determined that this provision needed to be more specific for three groups of systems: (1) Those performing Stage 1 DBPR reduced monitoring, (2) large ground water systems, and (3) small systems required to conduct an early IDSE. Today's proposal clarifies that these systems may use a 40/30 certification. EPA recognizes that these systems may have less compliance data on which to base their 40/30 certifications. However, EPA believes that the data that will be available are sufficient to make a determination on the most appropriate Stage 2B monitoring locations.

c. *Implementation.* Systems are required to submit an IDSE report so that primacy agencies may review the system's IDSE data collection efforts and the Stage 2B monitoring locations recommended by the system. Systems serving at least 10,000 must submit their IDSE report two years after rule promulgation (which may be prior to primacy for some States). The M–DBP Advisory Committee recommended an implementation schedule that would allow systems sufficient time to make site-specific risk determinations and decisions regarding the simultaneous implementation of the Stage 2 DBPR and LT2ESWTR but not stretch out the compliance time frame too far into the future. This provision requires that medium and large systems conduct and complete site-specific risk determinations (*i.e.*, the IDSE and LT2ESWTR *Cryptosporidium* monitoring) as soon as possible after rule promulgation. Since small systems cannot begin their microbial monitoring until after the results from the large system microbial monitoring have been analyzed, small systems have a longer compliance time frame.

Systems that submit a 40/30 certification are required to submit that certification as part of the IDSE report and to include a recommended Stage 2B monitoring plan. The monitoring plan is required for these systems because the Stage 2B MCL compliance monitoring sites proposed today have fundamentally different objectives than the Stage 1 DBPR monitoring sites. Additionally, many systems are required to have more Stage 2 compliance monitoring sites than Stage 1 sites because high HAA5 site may be different than high TTHM sites.

3. Request for Comment

EPA requests comments on the IDSE requirement and whether it is a good tool to identify sites representative of high TTHM and high HAA5 levels.

a. *Applicability.* EPA requests comment on requiring large (serving 10,000 or more people) nontransient noncommunity water systems to perform an IDSE. Should NTNCWSs serving fewer than 10,000 people be required to conduct an IDSE? EPA also requests comment upon whether States should be able to waive IDSE requirements for very small systems (serving fewer than 500 people). Are there objective criteria that the State should use in waiving the requirement? Should the State be allowed to grant very small system waivers based on some other criterion other than serving a population <500? For example, should the State be allowed to choose a higher population cutoff? Should the State be allowed to use a non-population criterion such as simplicity of distribution system to grant a very small system waiver? If so, what should this criterion be and how should qualification be demonstrated?

b. *Data collection.* EPA requests comment on the requirements for each of the alternatives for data collection under the proposed IDSE including: the standard monitoring program, the system-specific study, and the 40/30 certification. EPA requests comment on whether systems with less than two years of routine monitoring data should be considered to have sufficient data to utilize the 40/30 certification.

Specifically EPA requests comment on whether systems on reduced monitoring, large ground water systems, and small systems required to conduct an IDSE within the first two years after promulgation should be prohibited from submitting a 40/30 certification.

c. *Implementation.* EPA requests comment on the requirement that large and medium systems must collect data and prepare their IDSE report prior to State primacy. EPA requests comment from the States regarding whether they intend to be involved in the consultations with systems collecting data for IDSE or in the review of IDSE reports that are submitted prior to State primacy. EPA is developing a plan to implement the IDSE during the period prior to State primacy. EPA requests comment on any issues that should be addressed during this period to facilitate the IDSE.

I. Monitoring Requirements and Compliance Determination for Stage 2A and Stage 2B TTHM and HAA5 MCLs

1. What Is EPA Proposing Today?

Today's proposal includes new requirements for how systems must monitor TTHM and HAA5 levels in their distribution systems and how systems must assess their monitoring results to determine compliance with TTHM and HAA5 MCLs. The new monitoring requirements are associated with the IDSE (described in section V.H) and Stage 2B of the proposed rule. The new compliance determination requirements relate to use of the locational running annual average (LRAA) for meeting proposed Stage 2A and Stage 2B MCLs for TTHM and HAA5 (described in section V.D). This section presents these proposed monitoring and compliance determination requirements for Stage 2A, the IDSE, and Stage 2B.

An important aspect of the proposed TTHM and HAA5 monitoring requirements is the use of two different approaches for determining the number of samples a system is required to collect. One approach is plant-based. Under the plant-based approach, a system's TTHM and HAA5 sampling requirements are determined by the number of treatment plants in the system and, in the case of consecutive systems, the number of consecutive system entry points. The second approach is population-based. Under the population-based approach, a system's sampling requirements are influenced by the number of people served, but not by the number of treatment plants. EPA is proposing population-based sampling requirements only for IDSE and Stage 2B monitoring by consecutive systems that purchase all of their finished water year-round. However, EPA is requesting comment on applying a population-based approach to all systems for the IDSE and Stage 2B compliance. The discussion of monitoring requirements in this section provides details on these two approaches.

A number of factors affect DBP formation, including the type and amount of disinfectant used, water temperature, pH, amount and type of precursor material in the water, and the length of time that water remains in the treatment and distribution systems. For this reason, and because DBP levels can be highly variable throughout the distribution system (as discussed in section IV), today's proposal requires systems to collect IDSE and Stage 2B samples at specific locations in the distribution system and in accordance with a sampling schedule. For purposes of determining the number of required samples, EPA intends to maintain the provision in the Stage 1 DBPR (§ 141.132(a)(2)) that multiple wells drawing raw water from a single aquifer may, with State approval, be considered one plant, and prior approvals will remain in force unless withdrawn.

a. *Stage 2A.* For Stage 2A of the proposed rule, compliance will be based on the compliance sampling sites and frequency established under the existing Stage 1 DBPR. Systems must continue to monitor for TTHM and HAA5 using a plant-based approach, as required under 40 CFR 141.132. Using these monitoring results, systems must continue to demonstrate compliance with Stage 1 MCLs of 0.080 mg/L for TTHM and 0.060 mg/L for HAA5, based on a running annual average (see 40 CFR 141.133). In addition, systems must comply with the Stage 2A MCLs of 0.120 mg/L for TTHM and 0.100 mg/L for HAA5, based on the LRAA at each Stage 1 DBPR monitoring location. Stage 1 DBPR provisions for systems to reduce the frequency of TTHM and HAA5 monitoring will still apply.

Stage 2A will primarily affect surface water systems serving at least 10,000 people or systems with multiple plants, because these systems are required to monitor at more than one location in the distribution system. Most other systems take compliance samples at only one location under Stage 1 and in these cases, the calculated LRAA will be equal to the calculated RAA.

b. *IDSE.* IDSE monitoring requirements are designed to identify locations within the distribution system with high TTHM and HAA5 levels, which will serve as Stage 2B monitoring sites. The following discussion provides details on the IDSE standard monitoring program. Section V.H identifies other approaches by which systems can meet IDSE requirements of the rule.

For IDSE monitoring, subpart H systems serving at least 10,000 people must collect samples approximately every 60 days at eight distribution system sites per plant (these are in addition to Stage 1 DBPR compliance monitoring sites). The distribution system residual disinfectant type determines the location of the eight sites, as shown in Table V–3.

Subpart H systems serving fewer than 10,000 people and all ground water systems must collect IDSE samples at two distribution system sites per plant (at sites that are in addition to the Stage 1 DBPR compliance monitoring sites) as shown in Table V–3. Subpart H systems serving 500–9,999 people and ground water systems serving at least 10,000 people must sample quarterly (approximately every 90 days); subpart H systems serving fewer than 500 people and ground water systems serving fewer than 10,000 people must sample semi-annually (approximately every 180 days).

EPA is also proposing IDSE monitoring requirements for consecutive systems. For consecutive systems that both purchase finished water and treat source water to produce finished water, IDSE requirements are the same as for non-consecutive systems with the same population and source water type (*see* Table V–3). For these consecutive systems, each consecutive system entry point (defined in section V.C) is counted as one treatment plant for purposes of determining sampling requirements. However, the State may allow a system to consider multiple consecutive system entry points to be considered a single point.

As noted previously, for consecutive systems that purchase all of their finished water year-round, EPA is proposing a population-based monitoring approach (*see* Table V–4) instead of a plant-based approach. Under the population-based approach, monitoring requirements are not influenced by the number of consecutive system entry points, but are based solely on the population served and the type of source water used. EPA believes the population-based approach is equitable and will provide representative DBP concentrations throughout distribution systems.

TABLE V–3.—PROPOSED IDSE MONITORING REQUIREMENTS

System type and population served	Distribution system disinfectant type	Number of monitoring periods	Distribution system sample locations per plant per monitoring period [1]				
			Total	Near entry point	Average residence time	High TTHM locations	High HAA5 locations
Subpart H ≥10,000	Chloramines	[2]6	8	2	2	2	2
	Chlorine	[2]6	8	1	2	3	2
Subpart H 500–9,999 or Ground Water ≥10,000.	Any	[3]4	2	0	0	1	1
Subpart Any H <500 or Ground Water <10,000.	Any	[2]4	2	0	0	1	1
Consecutive Systems	Any	—Consecutive systems that purchase 100% of their finished water year-round—see Table V.4. —Consecutive systems that also treat source water to produce finished water—plant-based monitoring at same location and frequency as a non-consecutive system with the same population and source water.					

[1] Samples must be taken at locations other than the existing Stage 1 DBPR monitoring locations. Dual sample sets (*i.e.*, a TTHM and an HAA5 sample) must be taken at each site. Sampling locations should be distributed throughout the distribution system.
[2] Approximately every 60 days.
[3] Approximately every 90 days.
[4] Approximately every 180 days.

TABLE V–4. POPULATION-BASED MONITORING FREQUENCIES AND LOCATIONS UNDER IDSE FOR CONSECUTIVE SYSTEMS THAT PURCHASE 100% OF FINISHED WATER YEAR-ROUND

Source water type	Population size category	Monitoring periods and frequency	Distribution system sample locations [1]				
			Total	Near entry points [2]	Average residence time	High TTHM locations	High HAA5 locations
Subpart H	0–499	Two 2 every 180 days) ...	2	.....	.....	1	1
	500–4,999	Four (every 90 days)	2	.....	.....	1	1
	5,000–9,999		4	.....	1	2	1
	10,000-24,999	Six (every 60 days)	8	1	2	3	2
	25,000-49,999		12	2	3	4	3
	50,000-99,999		16	3	4	5	4
	100,000-499,999		24	4	6	8	6
	500,000-1,499,000		32	6	8	10	8
	1,500,000-4,999,999		40	8	10	12	10
	≥5,000,000		48	10	12	14	12
Ground Water	0–499	Two (every 180 days)	2	.....	.....	1	1
	500–9,999		2	.....	.....	1	1
	10,000-99,999	Four (every 90 days)	6	1	1	2	2
	100,000-499,999		8	1	1	3	3
	≥500,000		12	2	2	4	4

[1] Samples must be taken at locations other than the existing Stage 1 DBPR monitoring locations. Dual sample sets (*i.e.*, a TTHM and an HAA5 sample) must be taken at each site. Sampling locations should be distributed throughout the distribution system.
[2] If the number of entry points to the distribution system is less than the specified number of sampling locations, additional samples must be taken equally at high TTHM and HAA5 locations. If there is an odd extra location number, a sample at a high TTHM location must be taken. If the number of entry points to the distribution system is more than the specified number of sampling locations, samples must be taken at entry points to the distribution system having the highest water flows.

As a part of the monitoring schedule, all systems conducting IDSE monitoring must collect samples during the peak historical month for TTHM levels or water temperature. EPA will provide guidance to assist systems in choosing IDSE monitoring locations, including criteria for selecting high TTHM and HAA5 monitoring locations.

c. *Stage 2B.* For those systems required to conduct an IDSE, Stage 2B monitoring sites are based on the system's IDSE results and Stage 1 DBPR compliance monitoring results. For those systems not required to conduct an IDSE, Stage 2B monitoring locations are based on the system's Stage 1 DBPR compliance monitoring results and an evaluation of the distribution system characteristics to identify additional monitoring locations, if required.

Consistent with the Advisory Committee recommendations, the monitoring frequency for Stage 2B is structured so that systems that monitor quarterly under the Stage 1 DBPR will continue to monitor quarterly. In addition, the monitoring schedule must include the month with the highest historical DBP concentrations.

Many systems on reduced monitoring under the Stage 1 DBPR will conduct Stage 2B compliance monitoring at different or additional locations than those used for Stage 1 compliance monitoring. Such systems must conduct routine monitoring for at least one year before being eligible for reduced monitoring under Stage 2B. Those systems that monitor at the same locations under both the Stage 1 DBPR and Stage 2B DBPR and have qualified for reduced monitoring under Stage 1 may remain on reduced monitoring at the beginning of Stage 2B.